SMALL BOWEL GANGRENE ITS ETIOLOGY PATHOGENESIS MANAGEMENT AND ITS OUTCOME

Dissertation submitted in partial fulfilment of regulation for the award of

M.S. Degree in General Surgery (Branch I)

THE TAMILNADU

DR. M.G.R. MEDICAL UNIVERSITY

APRIL 2012

CERTIFICATE

Certified that this dissertation entitled **"SMALL BOWEL GANGRENE ITS ETIOLOGY PATHOGENESIS MANAGEMENT AND ITS OUTCOME"** is a bonafide work done by **Dr. R.MANIAPPAN**, post graduate student of the Department of General Surgery, Coimbatore Medical College, Coimbatore, during the academic year 2009 - 2012. This work has not previously formed the basis for the award of any degree or diploma.

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I solemnly declare that dissertation titled, **"SMALL BOWEL GANGRENE ITS ETIOLOGY PATHOGENESIS MANAGEMENT AND ITS OUTCOME"** is a bonafide work done by me at Coimbatore Medical College during 2009 - 2012 under the guidance and supervision of **Prof.Dr.G.RAVINDRAN M.S.,** Department of Surgery, Coimbatore Medical College, Coimbatore.

The dissertation is submitted to The Tamilnadu Dr. M.G.R. Medical University, towards partial fulfilment of requirement for the award of **M.S. Degree in General Surgery (BRANCH - I).**

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CONTENTS

INTRODUCTION

Small bowel gangrene is a major abdominal catastrophy associated with high mortality rate. This increased mortality rate is attributed to difficulty in diagnosing the condition early, the late presentation of the patient to the hospital and non availability of precise diagnostic tool for assessing bowel gangrene. Diagnosis is confirmed only during surgery.

Uncomplicated hernias are not given proper importance by the patient and presents to the emergency department with strangulated hernia. As most of the hernias occur commonly in old age , the co morbid conditions that are related to the age add up to the factors influencing the mortality rate of small bowel gangrene.

The important step toward reducing the mortality and morbidity of small bowel gangrene is early surgical intervention. Delay in intervention will lead to prolonged exposure of the patients to the lethal toxins and bacteria released by the gangrenous bowel and this will progress to irreversible hypotensive shock.

The most common cause of bowel gangrene secondary to mechanical obstruction is strangulated hernia in India and post operative adhesions in developed countries. The strangulated hernia can be prevented by early surgical intervention of uncomplicated hernias.

This study was conducted to analyze in detail the various etiology, common presentation, management and their outcome in small bowel gangrene and to discuss the ways in which the incidence of small bowel gangrene and its mortality rate may be reduced.

HISTORICAL REVIEW

1. **Sushrutha 6th** century B.C wrote the oldest known descriptions about bowel surgery. Described using a chemical cautery over the swelling of strangulated hernias. Used the tentacles of black ants to clamp the edges of bowel wounds together.

2. **Aretaeus the Cappadocian** (81-138 A.D.) Described in detail ileus secondary to incarcerated hernia.

3. **Fabricius d'Aquapendente** 12th century As reported by Duverger, he described a procedure of intestinal repair involving end-to-end anastomosis

4. **Lanfranc** 13th century Used animal tracheas to connect divided segments of bowel.

5. **Franco** 1556 described his experience in surgically treating strangulated inguinal hernia. He made an incision over the swelling, divided the constricting band, inserted a goose-quill-sized cannula, and returned the bowel to the peritoneum.

6. **Mery** 1701 Removed several feet of gangrenous bowel and established an artificial anus in a woman suffering from a strangulated hernia.

7. **Ramdohr** 1727 Removed two feet of gangrenous small bowel and invaginated the proximal end of the bowel into the lumen of the distal segment, securing the connection with a few sutures.

8. **Travers** 1812 While experimenting with suture techniques, he noted that wounds closed with sutures that passed through all layers of the bowel wall healed well.

9. **Jobert** 1824 Performed end-to-side anastomosis in dogs and cats using continuous wax suture.

10. **Lembert** 1826 Developed a suture technique employing interrupted sutures that passed through the entire bowel wall except for the mucous membrane.

11. **Schwartz** 1911 Used x-ray films to determine areas of intestinal distension

12. **Klass** 1950 Diagnosed mesenteric ischemia before infarction. Performed embolectomy without intestinal resection (patient died of acute heart failure).

13. **Shaw & Rutledge** 1957 Reported successful superior mesenteric vein embolectomy without bowel resection.

14. **Ende** 1958 First description of non occlusive mesenteric ischemia.

AIM OF THE STUDY

1. To study the various modes of presentation of small bowel gangrene.

2. To study the etiology of gangrene of small bowel in Coimbatore Medical College Hospital.

3. To study the outcome of the treatment.

4. To study the mortality and morbidity of small bowel gangrene.

5. To analyze the methods to reduce mortality and morbidity associated with gangrene of small bowel.

REVIEW OF LITERATURE

ANATOMY OF SMALL INTESTINE

The small intestine is the longest part of the alimentary canal and extends from the pylorus of the stomach to the ileocecal junction .It is divided into three parts: the duodenum, the jejunum, and the ileum.

DUODENUM

The duodenum is a C-shaped tube, about 25 cm long, which joins the stomach to the jejunum. It receives the openings of the bile and pancreatic ducts. The duodenum curves around the head of the pancreas . The first 2.5 cm of the duodenum resembles the stomach in that it is covered on its anterior and posterior surfaces with peritoneum and has the lesser omentum attached to its upper border and the greater omentum attached to its lower border; the lesser sac lies behind this short segment. The remainder of the duodenum is retroperitoneal, being only partially covered by peritoneum.Duodenum is divided into four parts.

First Part of the Duodenum

The first part of the duodenum begins at the pylorus and runs upward and backward on the transpyloric plane at the level of the first lumbar vertebra.

Second Part of the Duodenum

The second part of the duodenum runs vertically downward in front of the hilum of the right kidney on the right side of the second and third lumbar vertebrae .About halfway down its medial border, the bile duct and the main pancreatic duct

pierce the duodenal wall. They unite to form the ampulla that opens on the summit of the major duodenal papilla .The accessory pancreatic duct, if present, opens into the duodenum a little higher up on the minor duodenal papilla.

Third part of Duodenum

The third part of the duodenum runs horizontally to the left on the subcostal plane, passing in front of the vertebral column and following the lower margin of the head of the pancreas.

Fourth Part of the Duodenum

The fourth part of the duodenum runs upward and to the left to the duodenojejunal flexure .The flexure is held in position by a peritoneal fold, the ligament of Treitz, which is attached to the right crus of the diaphragm.

Blood Supply of duodenum

Arteries

The upper half is supplied by the superior pancreaticoduodenal artery, a branch of the gastroduodenal artery .The lower half is supplied by the inferior pancreaticoduodenal artery, a branch of the superior mesenteric artery.

Veins

The superior pancreaticoduodenal vein drains into the portal vein; the inferior vein joins the superior mesenteric vein .

Lymph Drainage

The lymph vessels follow the arteries and drain upward via pancreaticoduodenal nodes to the gastroduodenal nodes and then to the celiac nodes and downward via pancreaticoduodenal nodes to the superior mesenteric nodes around the origin of the superior mesenteric artery.

Nerve Supply

The nerves are derived from sympathetic and parasympathetic (vagus) nerves from the celiac and superior mesenteric plexuses.

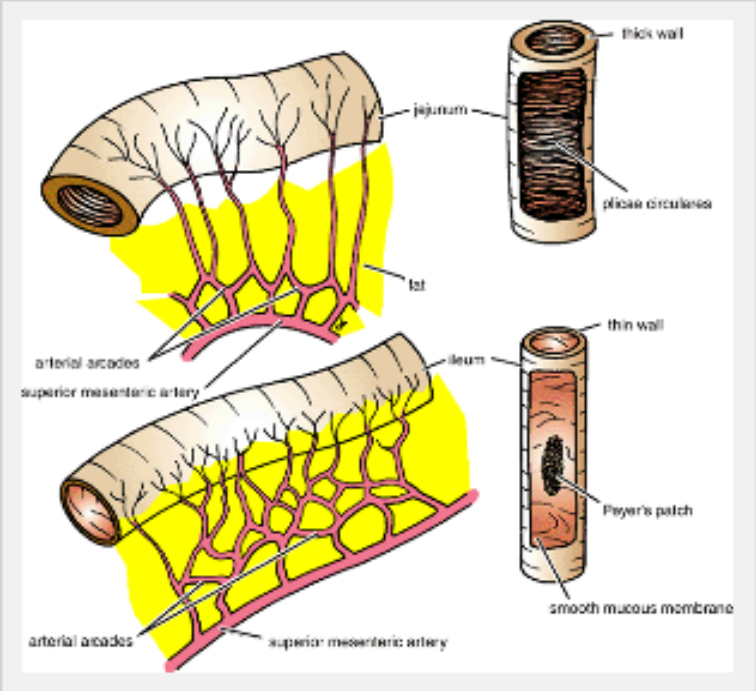
JEJUNUM AND ILEUM:

The jejunum and ileum measure about 20 ft (6 m) long; the upper two fifths is jejunum. Each has distinctive features, but there is a gradual change from one to the other. The jejunum begins at the duodenojejunal flexure, and the ileum ends at the ileocecal junction.

The coils of jejunum and ileum are freely mobile and are attached to the posterior abdominal wall by a fan-shaped fold of peritoneum known as the mesentery .The long free edge of the fold encloses the mobile intestine. The short root of the fold is continuous with the parietal peritoneum on the posterior abdominal wall along a line that extends downward and to the right from the left side of the second lumbar vertebra to the region of the right sacroiliac joint.

The root of the mesentery permits the entrance and exit of the branches of the superior mesenteric artery and vein, lymph vessels, and nerves into the space between the two layers of peritoneum forming the mesentery.

DIFFERENCE BETWEEN JEJUNUM AND ILEUM	
JEJUNUM	**ILEUM**
Thick wall	Thin wall
Large lumen	Small lumen
Fat on mesentery	Fat on ileum & mesentery
Prominent plicae circulars	Less prominent plicae
Single arterial arcade	Multiple arterial arcade
Sparse lymph node aggregate	Frequent lymph aggregate



Difference Between Jejunum and Ileum

Blood Supply

Arteries

Arterial supply by superior mesenteric artery.. They anastomose with one another to form a series of arcades. The lowest part of the ileum is also supplied by the ileocolic artery.

Veins

The veins correspond to the branches of the superior mesenteric artery and drain into the superior mesenteric vein .

Lymph Drainage

Through many intermediate mesenteric nodes reach the superior mesenteric nodes, which are situated around the origin of the superior mesenteric artery.

Nerve Supply

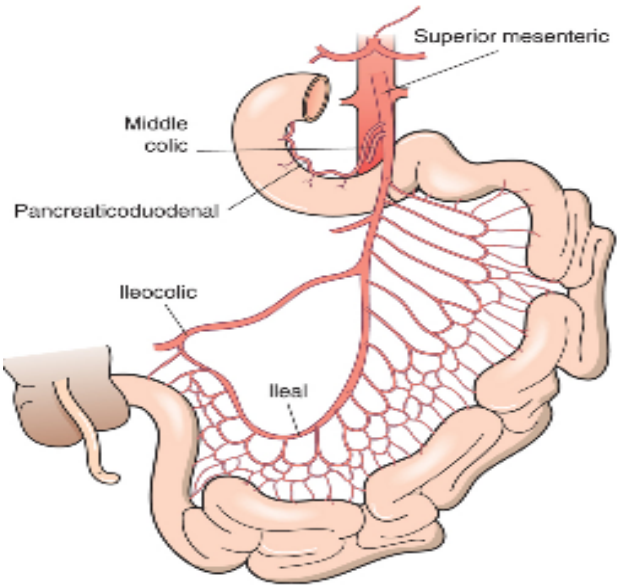
The nerves are derived from the sympathetic and parasympathetic (vagus) nerves from the superior mesenteric plexus.

SUPERIOR MESENTERIC ARTERY (SMA)

The SMA usually arises at a 20- to 30-degree angle from the anterior aspect of the aorta opposite the upper third of the body of L1, 5 to 15 mm caudal to the celiac artery. At its origin, the SMA measures about 1 cm in diameter. As it passes forward and downward, it emerges from beneath the inferior surface of the body of the pancreas and courses anterior to the third portion of the duodenum and uncinate process of the pancreas.

Constant branches of the SMA include the inferior pancreaticoduodenal, the middle colic, the right colic, the ileocolic, and the intestinal arteries. The inferior pancreaticoduodenal artery arises on the right side and communicates with pancreaticoduodenal branches from the gastroduodenal branch of the hepatic artery. The middle colic artery arises just distal to the inferior pancreaticoduodenal along the inferior border of the pancreas. This vessel is an important landmark when managing SMA occlusive problems. The right branch of the middle colic artery anastomoses with the ascending limb of the right colic artery and with the left branch of the middle colic artery, which comes from the inferior mesenteric circulation. The ileocolic artery may arise from the SMA either separately or in a common trunk with the right colic artery. Intestinal arterial branches supply the jejunum and ileum and vary from 12 to 20 in number. They originate from the left side of the SMA after it enters the mesentery.

Blood supply of small intestine by superior mesenteric artery



Branches of Superior mesentery artery

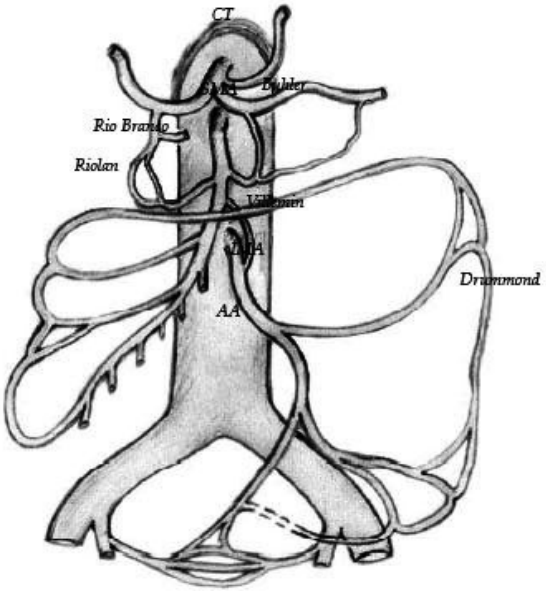


Figure shows branches of superior mesenteric artery and anastomosis of duodenopancreatic artery with both left colic and middle colic artery.

CELIAC-SUPERIOR MESENTERIC COMMUNICATIONS

A common origin of the SMA and the celiac artery occurs in 1% of individuals. In addition, between these two vessels may be a direct communicating channel, known as the **anastomotic artery of Bühler**, which is a remnant of an embryologic connection between these two arteries. Other important connections between individually arising superior mesenteric and celiac trunks are the superior and inferior pancreaticoduodenal arcades. Communication with middle colic arterial branches from the SMA occasionally occurs through the dorsal pancreatic branch of the splenic artery. After complete occlusion of the celiac axis, generous communications by the pancreaticoduodenal loop maintain hepatic and gastric circulations .

SUPERIOR MESENTERIC – INFERIOR MESENTERIC ARTERIES COMMUNICATION

The Meandering Mesenteric Artery connects the ascending branch of the left colic artery directly by a central anastomotic vessel to the SMA circulation with a branch arising from the SMA just proximal to the origin of the middle colic artery. The meandering mesenteric artery is potentially present in about two thirds of normal individuals.

The marginal artery of Drummond, first described by Von Haller in 1786 connects the left branch of the middle colic with the ascending branch of the left colic artery. At the splenic flexure, the left branch of the middle colic artery and the left colic artery from the IMA anastomose to provide continuity to the marginal artery of Drummond. This anastomotic site is Griffiths' point.

REGULATION OF MESENTERIC BLOOD FLOW

Active vasodilation results from lysis of basal intrinsic smooth muscle tone. Vasoconstriction results from changes in opposing constrictor and dilator forces favoring smooth muscle contraction. Changes in intestinal blood flow are influenced by numerous extrinsic and intrinsic factors operating simultaneously.

EXTRINSIC CONTROL

Autonomic nervous system

Sympathetic stimulation causes vigorous contraction of arteriolar smooth muscle resulting in significant reduction in intestinal blood flow. Redistribution of capillary perfusion may result from sympathetic stimulation of precapillary sphincters. The major physiologic role of sympathetic vasoconstriction of the gut is

to decrease splanchnic blood flow during activities that require increased blood flow to skeletal muscle, heart, and brain. Continued sympathetic discharge may cause persistent mesenteric vasospasm even after the underlying cause of intestinal hypoperfusion has been corrected. In addition, redistribution of blood flow away from the mucosa, mediated by sympathetic nervous activity may account in part for susceptibility of mucosa to ischemic damage in various pathologic conditions involving the mesenteric circulation.

Drugs

Norepinephrine, methoxamine, metaraminol, and phenylephrine produces predominant alpha-adrenoreceptor stimulation and constricts mesenteric vasculature. Alphablocking agents such as phentolamine cause intestinal vasodilation. Systemic administration of phentolamine may result in decreased intestinal blood flow even though local mesenteric vasodilation occurs.

Isoproterenol, a beta-adrenoreceptor stimulant, increases intestinal blood. This action is blocked by propranolol, a beta-antagonist. Epinephrine, which has both alpha- and beta-adrenoreceptor stimulating action, in low concentrations causes intestinal vasodilation, whereas higher concentrations produce vasoconstriction as the alpha effects dominate beta-mediated vasodilation. Dopamine causes vasodilation by stimulating dopaminergic receptors in mesenteric vessels. Dopamine levels greater than 10 μg/kg/min produce an alpha-stimulating effect that results in mesenteric vasoconstriction.

Histamine produces mesenteric vasodilation when it is administered intravenously or intra-arterially. Histamine causes contraction of nonvascular

intestinal smooth muscle, an effect that may limit the increase in intestinal blood flow produced by vasodilation. Bradykinin produces intestinal vasodilation.

Vasopressin and angiotensin II are peptides that produce potent vasoconstriction of the intestinal circulation selectively affecting the splanchnic resistance vessels. Vasopressin is released as a result of systemic hypotension, and if the hypotension results from mesenteric ischemia, vasopressin may exacerbate vasoconstriction in the mesenteric vessels.

Smooth muscle relaxant drugs causing vasodilatation are tolazoline and papaverine, sodium nitroprusside, sodium nitrite, caffeine and aminophylline.

Digoxin produces significant intestinal vasoconstriction and diminishes mesenteric blood flow.[26] Ergotamine causes increased vascular resistance .Prostaglandin E_1 stimulates formation of cAMP and causes vasodilation.

Gastrointestinal and pancreatic hormones

The synthetic analog of gastrin, pentagastrin, reduces mesenteric vascular resistance and increases intestinal blood flow. Cholecystokinin has been reported to produce both vasoconstriction and vasodilation under varying conditions.

INTRINSIC REGULATION

Metabolic Regulation

Conditions resulting in excessive oxygen demand relative to oxygen supply cause both accumulation of metabolites and diminished oxygen level in interstitial fluid and this produce relaxation of arteriolar smooth muscle and increase tissue perfusion. Oxygen supply and demand are thereby balanced.

Myogenic Regulation

Vascular smooth muscle tone is altered by arteriolar tension receptors in response to changes in transmural pressure. Increased vascular transmural pressure results in arteriolar vasoconstriction, increased vascular resistance, and diminished blood flow. Conversely, a decrease in transmural pressure causes vasodilation, diminished vascular resistance, and increased blood flow. The result of such regulation is maintenance of constant capillary pressure with minimal alterations in transcapillary fluid exchange. The myogenic control system is the principal factor in the protective mechanism termed autoregulation, which refers to the ability of the mesenteric circulation to maintain uniform total intestinal blood flow in the presence of widely varying systemic arterial blood pressures. Blood flow in intestinal villus vessels remains constant even when perfusion pressure is lowered from 100 mmHg to 30 mmHg.

SPECIAL ASPECTS OF MESENTERIC CIRCULATION

Reactive Hyperemia

Both metabolic and myogenic factors probably contribute to intestinal vascular dilation that characteristically occurs after cessation of brief periods of sympathetic stimulation or mesenteric arterial occlusion.The magnitude and duration of this reactive hyperemia are directly related to duration of decreased perfusion. The hyperemic response occurs uniformly throughout the bowel wall after an occlusion period of less than 1 minute. Increasing the duration of ischemia results in hyperemia localized predominantly to the muscularis layer.

Postprandial Hyperemia

During the initial phase of food intake, anticipation and ingestion of a meal are associated with increased mesenteric vascular resistance, probably caused by generalized sympathetic activity. In the second phase, digestion of food and absorption of chyme result in decreased mesenteric vascular resistance. This decreased resistance leads to an increase in superior mesenteric artery blood flow, which may be double resting blood flow. Decrease in iliac artery blood flow during digestion suggests redistribution of cardiac output to the mesenteric circulation at the expense of limb blood flow. At maximal blood flow during digestion, the small bowel receives most of the blood, 700 ml/100 g or more. The stomach receives 300 to 400 ml/100 g, and the colon receives 200 to 250 ml/100 g. Postprandial intestinal hyperemia correlates with the postprandial abdominal pain characteristically experienced by patients with chronic intestinal ischemia.

Autoregulatory Escape

Autoregulatory escape is a compensatory mechanism that accounts for maintenance of intestinal blood flow at nearly normal levels, even in the presence of vasoconstrictor influences of continued sympathetic activity or prolonged catecholamine stimulation. Autoregulatory escape probably occurs because local metabolic vasodilator mechanisms, elicited by ischemia, become predominant over continued sympathetic vasoconstriction. Subsequent reversal of effects of vasoconstriction and restoration of required blood flow are greater in submucosa than in mucosa. Therefore, there may be a relative redistribution of blood flow away from mucosa. This phenomenon may explain preferential ischemic damage to the mucosa.

Mucosal Countercurrent Exchange Mechanisms

The architecture of the microcirculation in the intestinal villus accounts for the existence of a countercurrent exchange mechanism. Blood flow in the central part of the villus arteriole is parallel but opposite in direction to that in the subepithelial venous capillaries. Consequently, a gradient in oxygen tension exists between arteriole and venule. This gradient is most prominent at the base of the villus. Diffusion along the gradient results in a progressive decrease in oxygen tension as blood flows from the base to the tip of the villus. This progressive diffusion gradient is accentuated by conditions that cause low mesenteric flow rates. The counter-current exchange mechanism, therefore, tends to aggravate tissue hypoxia in ischemic conditions of the bowel.

Collateral Blood Flow

When a major mesenteric artery becomes occluded, diminished arterial pressure distal to the obstruction stimulates collateral pathways to open promptly. Blood flow through collateral vessels continues as long as pressure in the vascular bed distal to the occlusion remains lower than systemic pressure. Likewise, if the major vessel occlusion is corrected, blood flow through collateral channels ceases.

Intestinal Intraluminal Pressure

During an ischemic insult the intraluminal pressure increases and this explain the colicky abdominal pain associated with early bowel ischemia. Bowel distension caused by increased intraluminal pressure may diminish blood flow to the involved segment or to the entire intestine. An associated shunting of blood occurs away from the mucosa and muscularis propria. The well-perfused serosa imparts a normal pink

external appearance to the bowel, even though total intestinal blood flow may be markedly reduced. Diminution in flow may persist for hours after relief of distension. These findings emphasize the importance of nasogastric suction for decompression of the bowel in managing intestinal ischemia.

Response to Ischemia

Ultrastructural changes in mucosal cells are evident within 10 minutes of acute superior mesenteric artery occlusion, and extensive histologic changes occur within 30 minutes. Progression of these changes ultimately results in bowel necrosis . Important consequences of bowel ischemia are increased transcapillary filtration, interstitial edema, and ultimately the net movement of fluid into the bowel lumen. The enzyme xanthine oxidase reacts with hypoxanthine, which accumulates because of catabolism of ATP, and molecular oxygen to produce the cytotoxic oxygen radicals. The cytotoxic effects of the oxygen radicals presumably result from peroxidation of the lipid components of cellular and mitochondrial membrane.

Polymorphonuclear leukocytes appear to contribute to reperfusion injury by releasing lysosomal enzymes at the site of ischemic injury and by production of oxygen free radicals through neutrophil nicotinamide adenine dinucleotide phosphate oxidase.

ETIOLOGY OF SMALL BOWEL GANGRENE

1. **MAJOR VASCULAR OCCLUSION**

Thrombosis	Systemic atherosclerosis
Embolism	Recent myocardial infarction
	Congestive cardiac failure
	Arrhythmias
	Rheumatic fever
Non occlusive	
Mesenteric ischemia	Cardiogenic shock
	Cardiogenic bypass
	Vasopressor agent
	Sepsis ,burns, pancreatitis
Mesenteric vein	
Thrombosis	Hyper coagulability
	Pulmonary hypertension
	Inflammation, prior surgery, trauma

2. **BOWEL OBSTRUCTION**

Carcinoma, adhesions, strangulated hernia, stricture, diverticluar disease, volvulus

3. **SMALL VESSEL DISEASE**

Diabetes mellitus, rheumatoid arthritis, systemic lupus erthematosis, systemic vasculitis disorder, amyloidosis, radiation injury

4. MEDICATION

Diuretics, digitalis, oestrogen, danazol, NSAIDS

5. **HAEMATOLOGICAL DISEASE**

Sickle cell disease, protein C and S deficiency, anti thrombin III deficiency.

PATHOPHYSIOLOGY

The earliest ultrastructural changes are noted in the mucosal layer as soon as 10 minutes after injury . Histologic changes follow with the inflammatory cell infiltration. Bowel wall edema ensues as a result of loss of capillary integrity. Absence of this natural barrier permits bacterial translocation, promotion of endotoxemia, as well as exudation of fluid into the bowel lumen. The injured mucosa sloughs, leaving ulcerations of the bowel wall. Although the bowel may still be viable when the mucosa is threatened, prolonged interruption of blood flow ultimately leads to necrosis of the muscularis and serosa, a point at which the compromised segment is no longer salvageable.

In mesenteric vein thrombosis (MVT) there is a rise in portal and superior mesenteric venous pressures. In the intestine, increased hydrostatic pressure leads to luminal fluid sequestration as well as bowel wall edema. The ensuing relative hypovolemia and hemoconcentration may contribute to vasoconstriction. Ultimately, infarction of the affected intestinal segments may develop. Eventual focal

haemorrhage and necrosis lead to loss of the gut barrier function, which ultimately allows for bacterial translocation and possible endotoxemia. The arterial response to mesenteric venous thrombosis may persist well after the venous obstruction has been corrected.

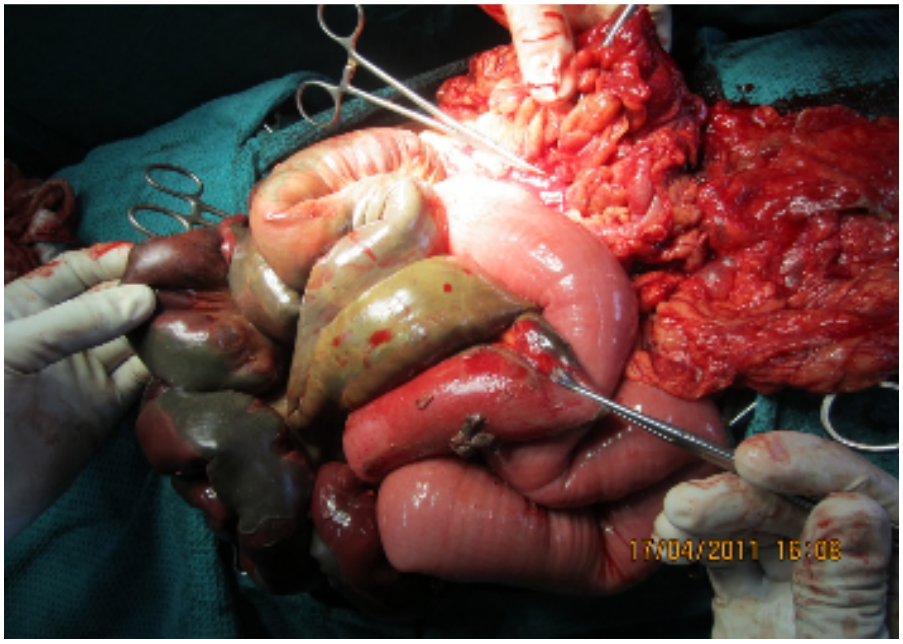
PATHOLOGY

GROSS APPEARANCE

Bowel turns to black in colour. Mucosa is denuded exposing the muscle fibres. The fibres are destroyed showing haemorrhagic patches and some time perforation of bowel wall.

MICROSCOPIC APPEARANCE:

Acute inflammatory cell are seen in plenty. Mucosal and sub mucosal edema present. Thrombi in mucosal and submucosal capillaries are characteristics. Muscle fibres shows loss of nuclei . muscularis propria is spared.



Intra Operative picture of small bowel gangrene

Resected segment of small bowel gangrene

CLINICAL PRESENTATION:

Patients present with severe generalised abdominal pain. Pain will often precede emesis. The location of pain may vary but as ischaemia progresses to infarction, the patient will develop generalised peritonitis. Patients can also present with symptoms of nausea, vomiting and diarrhoea early in the course of the disease. Progression of intestinal ischaemia to transmural bowel infarction may be signalled by fever, bloody diarrhoea and shock.

Early diagnosis of acute mesenteric ischemia (AMI) requires a high index of clinical suspicion in any patient who are at high risk for embolic or thrombotic events. Patients at particularly high risk include those with cardiac disease, peripheral vascular disease, cardiac arrhythmias or recent history of myocardial infarction. Physical examination of the abdomen may reveal relatively normal findings or only

slight abdominal distension in the early stages of AMI. As the disease progresses from bowel ischaemia to transmural bowel infarction, the abdomen becomes grossly distended with absent bowel sounds and peritoneal signs will develop.

EVALUATION OF INTESTINAL BLOOD FLOW

1.PHYSICAL SIGNS AND SYMPTOMS

It is rapid and inexpensive but has low specificity and low sensitivity.

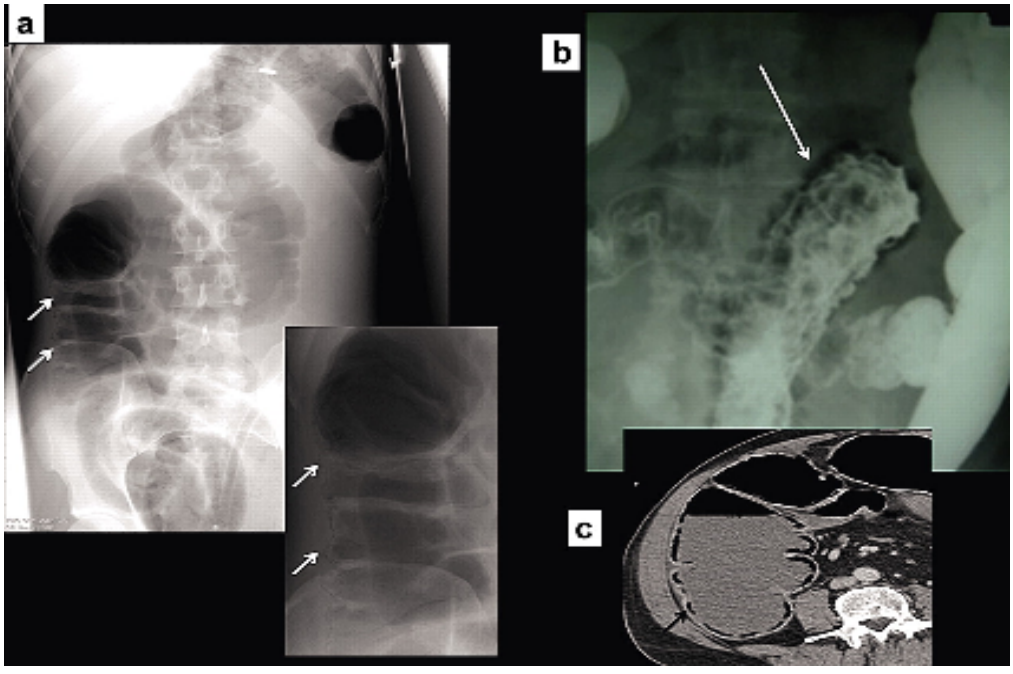
2.LAB INVESTIGATIONS

Elevated WBC count, amylase level, pH level are seen in bowel ischemia but has low specificity. Elevated venous and peritoneal phosphate level and eleveated creatinine phosphokinase are also elevated but many prospective study shows that it could not find difference between viable and non viable bowel.

3.PLAIN X RAY ERECT ABDOMEN

Gasless abdomen caused by small bowel spasm with subsequent distension and ileus but the disadvantage is that its non specific. Portal venous gas is a terminal finding and not use in early diagnosis. On barium examination thumb printing is seen due to submucosal haemorrhage and superficial mucosal ulceration.

X ray showing portal venous gas



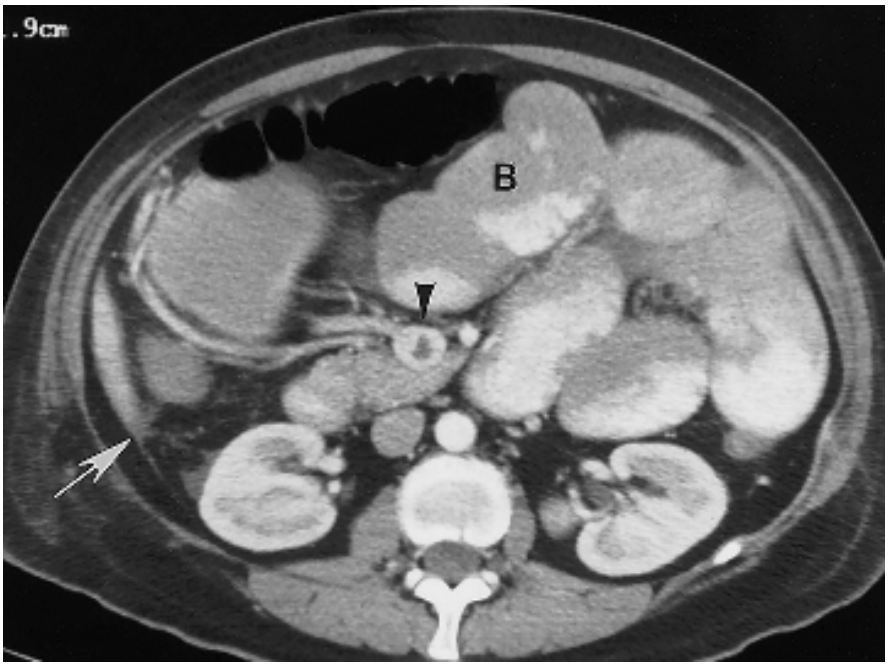
X ray showing intramural gas shadow

X ray showing thumb printing sign in transverse colon

4.COMPUTER TOMOGRAPHY OF ABDOMEN:

CT has become the diagnostic modality of choice in acute mesenteric vein thrombosis (MVT), with sensitivity exceeding 90%. The superior mesenteric or portal vein appears enlarged, with a central area of low attenuation, suggestive of thrombus. In the contrast phase, a rim may enhance at the vein wall, yielding a bull's eye appearance. Bowel wall thickening and the presence of ascites are also suggestive of the diagnosis of MVT. It is not helpful to differentiate between acute and chronic occlusion.

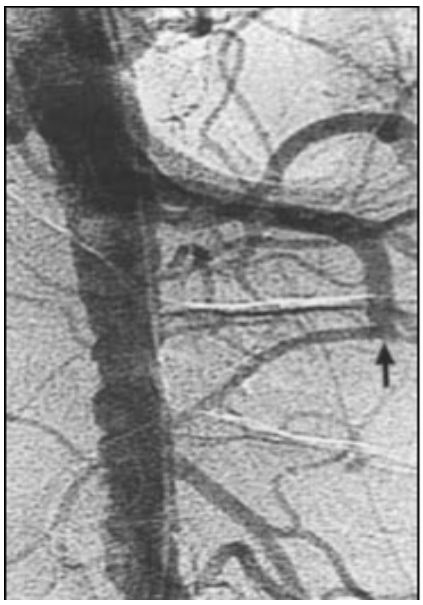
CT – abdomen shows block in superior mesentery artery



5.ARTERIOGRAPHY

Acute thrombosis involve origin of any or all the three vessels i.e. superior mesenteric, inferior mesenteric and celiac artery. Emboli affects mainly superior mesenteric artery often at the orifice of middle colic artery and visualized as an inverted meniscus sign.

In non occlusive ischemic disease segmental mesenteric arterial constriction with associated proximal stenosis of superior mesenteric artery. Anteriolateral and anterioposterior view is essential. Diagnostic information afforded by angiography allows the surgeon to select the appropriate operative approach. Typically, narrowing at the origin of the major SMA branches, or intermittent areas of narrowing and dilation ("string of sausages" or "string of lakes" sign), is seen.



Arteriogram showing Superior Mesentery Artery Occlusion

6. RADIOLABELLED ISOTOPE STUDIES

Intravenous **Te^{99m} labelled pyrophosphate** binds to extravasated extracellular calcium from ischemic cells and **Te^{99m} sulphur colloid labelled leucocyte** migrate to area of inflammation. Major disadvantage is that they need advanced bowel ischemia i.e necrosis to obtain hot spot in isotope imaging.

Intra peritoneal injection of Xe^{133} : Injected Xe^{133} quickly and equally absorbed by passive transperitoneal diffusion into ischemia and normal bowel . It is promptly cleared by normally perfused bowel and the poorly perfused bowel retain Xe^{133} which is detected by gamma cameras. The advantages are its safety, rapidity of results, ability to detect early ischemia, lack of interruption by adhesions and moderate ascites.

7. DOPPLER STUDY

Pre operative: It is valuable in evaluation of chronic mesenteric arterial occlusive disease. Furthermore, even if flow is seen in the proximal SMA or celiac arteries, an embolic etiology is not necessarily excluded. Absent flow in the portal and mesenteric venous system and the presence of ascites are highly suggestive of Mesenteric vein thrombosis. Transgastric ultrasonography has been suggested as a means of visualizing the visceral aorta.

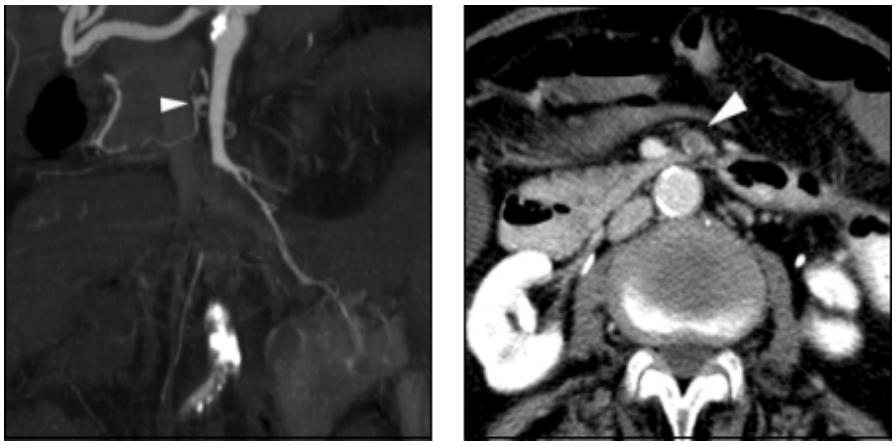
Intra operative : pencil like hand held Doppler ultrasound probe applied to the anti mesenteric border of bowel wall and discrete mesenteric vessels. Its shows typical pulsatile sound indicating adequate blood flow. Draw back is lack of sensitivity for small patchy areas of inadequate circulation and no use in venous occlusion. Advantage is its quickness and ease of application. Sensitivity is 82%and specificity is 91%.

8. FLOURESCEN DYE

Sodium flourescen dye emits gold-green flourescen when exposed to UV light of wave length 3600°A to 4000°A. It is injected intravenously over 30-60 seconds. The dye enters into the viable tissue with in minutes of injection. After dimming the operation room lights, woods lamp used to illuminate the field. The viable bowel show confluent fluorescence and non viable bowel show absence of florescence and perivascular fluorescent pattern. Its rapid transudation into peritoneal cavity and its prolonged presence after injection prevent it from using frequently atleast with in 48 hours. Over all sensitivity is 100% and sensitivity is 100%.

9. MAGNETIC RESONANT ANGIOGRAM

MRA are excellent non invasive screening techniques for patients suspected of having mesenteric ischemia of all causes. MRA has higher spatial resolution, allowing assessment of the peripheral visceral branches and the inferior mesenteric artery with greater accuracy. In addition, it allows the identification of calcified plaques. The lack of radiation and iodinated contrast agents make it the technique of choice for children and patients with azotemia.



MR angiogram showing superior mesentery artery block

10. MAGNETIC RESONANT OXIMETRY

MR oximetry is capable of detecting oxygen desaturation caused by segmental ischemia. A loss of oxygen saturation in the SMV relative to that in the inferior vena cava provides a convenient marker of mesenteric ischemia.

MANAGEMENT

Aggressive fluid resuscitation is vital and can be guided by placement of a urinary drainage catheter as well as a central venous or Swan-Ganz catheter in the patient with significant cardiac disease. An arterial line is indicated for systemic blood pressure monitoring if significant hemodynamic instability is noted. Dopamine is a more appropriate inotropic agent among patients with AMI because, in low doses, it may act as a mesenteric vasodilator, and in higher doses, it produces less severe mesenteric vasoconstriction than the latter agents. A nasogastric tube should be placed to decompress the fluid-filled and distended intestinal tract and thus promote intestinal perfusion, reduce the risk for bowel perforation, and minimize the chance of aspiration. In view of the potential for bacterial translocation through the compromised intestinal barrier, early institution of broad-spectrum antibiotics, including anaerobic coverage, is mandatory. Postoperatively, therapeutic anticoagulation is indicated in patients who have experienced an embolic occlusion to minimize the risk for recurrent embolization to the mesenteric circulation or other arterial beds.

INTERVENTIONAL RADIOLOGY

The primary treatment of (Non Occlusive Mesenteric ischemia) NOMI is pharmacologic. Specifically, selective catheter-directed administration of a number of vasodilating agents, including papaverine, tolazoline, glucagon, nitroglycerin, nitroprusside, prostaglandin E, phenoxybenzamine, and isoproterenol, have been used. A selective Ssuperior mesenteric artery test injection of papaverine, 60 mg, should be administered, followed by a repeat contrast injection. If this demonstrates reversal of vasoconstriction, the catheter is left in place, and a continuous infusion at

30 to 60 mg/hour is delivered. Side effects of papaverine include cardiac arrhythmia , hypotension ,reflex tachycardia. The follow-up angiogram should be repeated only after flushing the drug out of the infusion line for 30 minutes with saline. Based on the angiographic findings and the patient's clinical course, the infusion may be stopped or continued for an additional 24 hours. Infusions continued for as long as 4 and 5 days. Failure to improve or any evidence of a deteriorating clinical state mandates immediate surgical exploration. Other interventional radiologic techniques used in recent years in the management of AMI include catheter-directed thrombolysis, percutaneous transluminal angioplasty (PTA), and fenestration of aortic dissection.

SURGERY

Operative intervention remains the mainstay of management . The surgeon's goal is to confirm the diagnosis of mesenteric ischemia and assess bowel viability, determine the responsible etiology, perform revascularization where possible, and resect nonviable bowel.The presence of a strong pulsation in the proximal vessel, which weakens or is not palpable more distally, is highly suggestive of an embolus, whereas an absent pulsation in the proximal SMA is most consistent with arterial thrombosis. The continuous-wave Doppler should be used to detect flow if no pulses are palpable. The presence of a pulse or attenuated Doppler signal at the SMA origin, but with an absent signal in the mesentery, implies embolic occlusion of the vessel. Similarly, the celiac axis and its main branches should be examined.

REVASCULARISATION

EMBOLUS

The SMA should be controlled just distal to the origin of the middle colic artery and proximal to the jejunal branches and an arteriotomy performed. If the diagnosis of embolus is certain, we prefer a transverse arteriotomy, although if there is any doubt, a longitudinal incision should be made so that it can serve as the site for the distal anastomosis of a bypass graft. Not infrequently, the embolus can be localized at and directly extracted from this site. The thromboembolectomy catheter is passed proximally and distally to retrieve the embolus and associated thrombotic material .

THROMBOSIS

Thromboendarterectomy procedures is done on suprarenal aortic and proximal mesenteric arterial exposure. In acute SMA thrombosis, the distal anastomosis can usually be performed at the level of the middle colic artery or just proximal or distal to this. If celiac axis occlusion present, a distal anastomosis may be performed to the vessel just beyond its origin from the aorta or to any of its major branch vessels. When MVT is confirmed at exploration, although the primary treatment for this condition is anticoagulation, if there appears to be clot in the superior mesenteric vein, thrombectomy should be attempted.

RESECTION

After revascularization, 30 to 45 minutes of observation should be allowed before making a definitive assessment of intestinal viability and the necessity for bowel resection. Clinical signs, such as absence of peristalsis, bowel wall edema,

discoloration of the bowel and mesentery, mucosal haemorrhage, and absence of bleeding from cut bowel edges, are imprecise markers and may lead to resection of intestinal segments. A continuous-wave Doppler ultrasound probe or sodium fluorescein dye can be used to assess the viability of the bowel. All bowel judged to be nonviable must be resected. Primary anastomoses can be performed if brisk bleeding from the edges of the bowel wall is observed and the patient is stable. Alternatively, long segments of marginal bowel left in situ may be stapled or oversewn, with continuity established during a second-look procedure.

STRANGULATED INGUINAL HERNIA

A hernia is said to be strangulated when the blood supply of its contents seriously impaired due to constricted neck of the sac. Gallegos and associates estimated the probability of strangulated hernia over time to be 2.8% over 3 months and 4.5% at 2 years because of enlargement of the neck of the sac.

PATHOLOGY

The venous return is first impeded and the intestine becomes congested and bright red. As the venous stasis increases the arterial supply is also impaired. Ecchymosis appears in the serosa. Blood comes out into the intestine lumen and into the hernia sac. The vitality of the intestine diminishes and migration of bacteria through the intestinal wall and the fluid within the sac becomes full of bacteria and toxins.the mesentery within the sac becomes congested and haemorrhagic . Thrombosis of its vessel occurs. Gangrene starts 5-6 hours after the onset of first symptom of strangulation.

In strangulated hernia with onset of obstruction, gas and fluid accumulate within the intestinal lumen proximal to the site of obstruction. The intestinal activity increases in an effort to overcome the obstruction, accounting for the colicky pain and the diarrhoea. Most of the gas that accumulates originates from swallowed air, although some is produced within the intestine. The fluid consists of swallowed liquids and GI secretions .With ongoing gas and fluid accumulation, the bowel distends and intraluminal and intramural pressures rise. The intestinal motility is eventually reduced with fewer contractions. The luminal flora of the small bowel changes and a variety of organisms have been cultured from the contents. If the intramural pressure becomes high enough, intestinal microvascular perfusion is impaired, leading to intestinal ischemia, and, ultimately, necrosis.

CLINICAL FEATURES

Abdominal pain and vomiting is the main features. Pain is first at the hernia site and later spread all over the abdomen. If the strangulation is not relieved the paroxysm of pain continues. Such pain will cease only with onset of gangrene and paralytic ileus. On examination the hernia is tense and tender. Cough impulse is absent

Picture of Strangulated left inguinal hermia

Picture of Strangulated Richter's Hernia

STRANGULATED RICHTER'S HERNIA

When a portion of the circumference of the intestine becomes the content of the sac it is called Richter's hernia. Strangulation of such hernia often complicates a femoral hernia.

Clinical features mimic gastroenteritis and diagnosis becomes difficult. Vomiting if present is not that frequent. Intestinal obstruction may not develop until half the circumference of the bowel is involved. Intestinal colic may occur and there may be even diarrhoea. Absolute constipation occurs when the paralytic ileus sets in. For above said reasons diagnosis becomes delayed and operation performed mostly only when the gangrene and peritonitis sets in.

STRANGULATED FEMORAL HERNIA

In femoral hernia the contents pass through the femoral ring, transverse the femoral canal and comes out through the saphenous opening. After this it progress upwards in the subcutaneous tissue of the thigh and may even reach above the inguinal ligament. Due to the narrow passage of the canal,femoral hernia is more liable to strangulation. Its more common in females and accounts for 20% of hernia cases.

CLINICAL FEATURES

In case of strangulated hernia patient develops sudden pain at the local site which immediately spread all over the abdomen. Swelling is seen below and lateral to the pubic tubercle and below the inguinal ligament. Globular in shape and tense and tender on palpation.

STRANGULATED EPIGASTRIC HERNIA

When the hernia protrude in the midline through the interlacing fibres of the linea alba between umbilicus and xiphisternum it is called as epigastric hernia. These hernia begins as a protrusion of extra peritoneal fat and that is why it also called as **'fatty hernia of the linea alba'.**

AETIOLOGY

Sudden strain help to bring out epigastric hernia. Such strain will lead to tearing of the interlacing fibres of linea alba. These hernia is mostly restricted to young muscular manual workers.

CLINICAL FEATURES

It is often asymptomatic and gets pain when the contents get strangulated. In partial strangulation the pain often gets worse on physical exercise and on wearing tight dress. On examination the swelling feels firm, doesn't have cough impulse and cannot be reduced. Its tender and warm.

STRANGULATED VENTRAL HERNIA

It is also known as incisional hernia or post operative hernia. This occurs through an acquired scar in the abdominal wall caused by previous surgery.

ETIOLOGY

1. Patient related factor

Obese individual with lax muscle, patients suffering from chronic cough, patient with severe anaemia, hypo proteinemia or vitamin c deficiency.

2.Operative fallacies

Injury to the motor nerves supplying the area can occur during incision. Certain incision are vulnerable to cause nerve injury for example Kocher's subcostal incision injuries the 8,9,10th intercostal nerves, Battle's pararectal incision for appendicectomy may injure subcostal or ilioinguinal nerve. Inadequate care in wound closure and haemostasis. Keeping tube drainage through the laparotomy wound.

3.Postoperative causes

Infection, post operative cough and distension, post operative peritonitis, early removal of sutures, steroid therapy in post operative period.

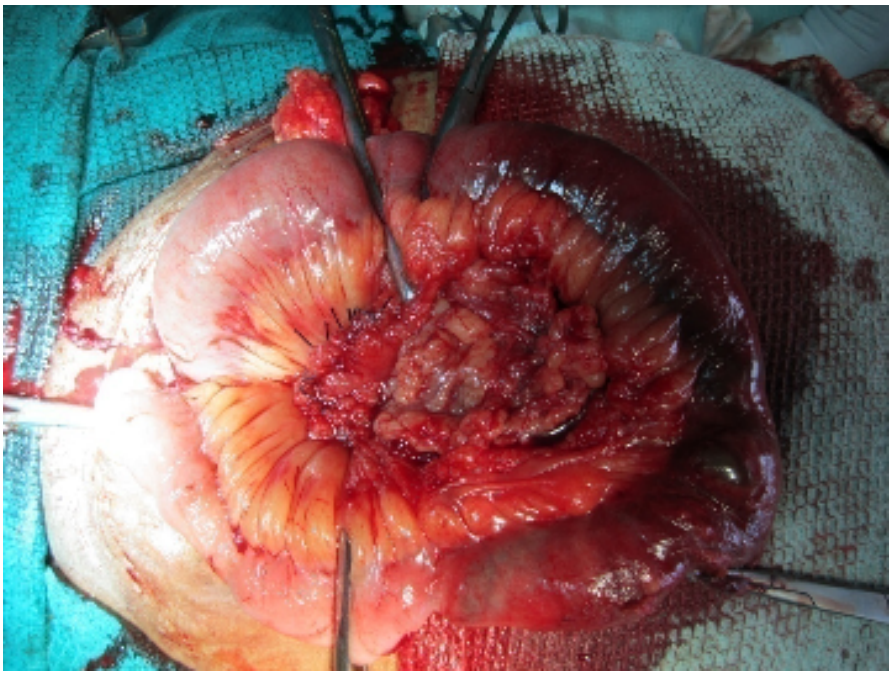
PATHOLOGY

Often the incisional hernia starts unnoticed and symptomless with partial disruption of the deeper layers of laparotomy wound during immediate or early post operative period. So careful closure of the wound is extremely important. Further wound infection often causes disruption of sutures thus the muscle are separated by weak scar tissue. A portion of the muscle may also be destroyed and heals by fibrosis which forms a weak scar.

CLINICAL FEATURES

Previous operation or a trauma is usually noted. Ventral hernia can occur at any age, but more common in fatty elderly females. Commonly presents as swelling and pain with features of intestinal obstruction. Strangulation, though uncommon, is liable to occur at the neck of small sac or in locule of a large hernia.

The old scar is seen with in the swelling. The hernia may occur through a small portion often at the lower end. The swelling is irreducible and cough impulse absent and its tense and tender.



Intraoperative picture of strangulated ventral hernia

TYPES OF VENTRAL HERNIA

Type 1: This hernia is situated in the midline. There is wide gap in musculature which is easily palpable. Mostly it is reducible and risk of strangulation is almost negligible.

Type 2: Situated in the lateral part. The defect is in the musculature and it is relatively small and irregular. Content are bowel and omentum usually matted together and adherent to the loculated peritoneal sac. So this hernia is partially or wholly irreducible and the risk of strangulation is high .

TREATMENT OF STRANGULATED HERNIA

Emergengy operation is treatment of choice to save patient's life. Initial management include :

1. Intra venous fluid administration. Foleys catheterisation done.
2. A nasogastric tube is inserted as intestinal obstruction is always associated.
3. Parenteral antibiotics is started immediately.
4. Taxis and reduction of hernia sac should be avoided as it will result in reduction-en-masse, rupture of intestinal wall, rupture of sac extra peritoneally.

Incision and opening of the sac is done according to the site of the hernia. The constricting narrow neck is divided. Viability of the bowel is the main thing to be considered . The following points indicate non viable bowel:

1. Bowel becomes greenish or blackish in colour.
2. It becomes flaccid and lustreless with thrombosis of the mesenteric vessels.
3. No peristalsis in the gut
4. Blood stained or foul smelling fluid in the sac.

It is always advisable that hot wet mops are placed on the involved bowel for 10 minutes and again watched for viability. If the loop is gently pinched with forceps peristaltic movement starts and returning of pink colour indicates returning of viability.

If the bowel is viable it is pushed back into the peritoneal cavity and herniorrhaphy done. No form of hernioplasty should be attempted. If the bowel is non viable (i) a linear patch of gangrene at the constriction ring is best treated by invaginating it by means of Lembert's suture. (ii) when the whole loop of the bowel is gangrenous and the condition of the patient permits resection anastomosis should be done. If the patient condition is poor and the bowel above the strangulation is grossly distended it is better to exteriorize the bowel and once the patient becomes fit restoration of the continuity is attempted. Closure of the hernia defect done according to the hernial site.

POSTOPERATIVE ADHESIONS

Adhesions may be defined as abnormal connective tissue attachments between tissue surfaces. It can be congenital or acquired (post inflammatory and postoperative). Postoperative adhesions are the leading cause of small bowel obstruction in Western societies and are responsible for 40–80% of bowel obstructions .The inciting trauma triggers an inflammatory response leading to activation of the complement and coagulation cascades, along with exudation of fibrinogen rich fluid .It appears that adhesions form in response to the initial fibrin gel matrix combined with the local microenvironment. If the fibrin gel allows apposition of adjacent surfaces, a band or bridge may form. This process of adhesion formation also is a dynamic process, consisting predominantly of macrophages early on, but by 2–4 days, larger strands of fibrin and fibroblasts begin to appear. By 5 days, distinct bundles of collagen are apparent, and fibroblasts begin to form a syncytium within the matrix. These cells thereafter predominate, and eventually the fibrin matrix and cellular elements are replaced by a vascularised, granulation-type tissue containing

macrophages, fibroblasts, giant cells, and a rich vascular supply. Eventually the surface of the adhesion are covered by a mesothelial layer, but only after formation of the underlying fibrous scar leading to surface opposition and transperitoneal bands.

The operations most frequently associated are colonic, rectal, and gynecologic procedures. Adhesive bowel obstruction may occur at any time postoperatively after a laparotomy, with reports ranging as early as within the first postoperative month to more than 8 decades after the index operation. Other causes include post peritonitis, post irradiation, intraperitoneal chemotheraphy, trauma to viscus and peritoneum, ischemia following ligatures, foreign bodies like gauze piece,glove powder, starch.

SMALL BOWEL VOLVULUS (SBV)

ETIOLOGY AND PATHOGENESIS

The causes of SBV may be classified as either primary or secondary.

Primary Small Bowel Volvulus

This occurs in an otherwise normal abdominal cavity and is much more common in Africa and Asia. Although the aetiology is still poorly understood, several aetiological factors have been proposed.

Diet. They suggested that diet may be a factor as the patients had eaten large quantities of fibre after prolonged fasting.

Gut motility. Increased intrinsic gut motility has a role in the aetiology of SBV. Parasitic infestation is known to alter small bowel motility, high concentrations of 5 hydroxy-tryptamine (5HT), a known stimulant of gut motility. Diabetic autonomic neuropathy may contribute to some cases of SBV.

The suggested mechanism underlying primary SBV is that a bulky bolus of food enters the proximal jejunum, causing the loop to descend into the pelvis. This displaces empty small bowel loops upwards, initiating rotation of the mesentery and leading to volvulus. For this to occur, there must be a combination of a long small bowel attached to a broad-based, fat-free mesentery (which splints the bowel), very firm abdominal muscles (restricting bowel movement to the coronal plane) and a diet with an exceptionally high bulk, eaten rapidly on an empty stomach.

Secondary Small Bowel Volvulus

The most frequently related conditions are bands, adhesions, Meckel's diverticulum, internal hernia, Ascariasis, pregnancy , ileal atresia, meconium ileus, enteroenterostomy, leiomyoma of the mesentery, and following operations, particularly gastrostomy, gastrectomy, and total hip replacement. The suggested mechanism of secondary volvulus involves obstruction of a small bowel loop at two fixed points by one of these predisposing conditions. As the loop fills with liquid, peristalsis causes it to twist around its mesentery.

PATHOPHYSIOLOGY:

In volvulus , a dangerous form of bowel obstruction called closed loop obstruction occurs, in which a segment of intestine is obstructed both proximally and distally. In such cases, the accumulating gas and fluid cannot escape either proximally or distally from the obstructed segment, leading to a rapid rise in luminal pressure, and a rapid progression to strangulation.

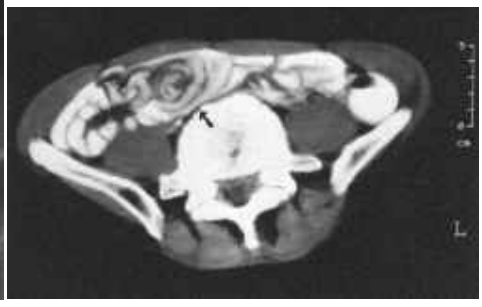
CLINICAL FEATURES

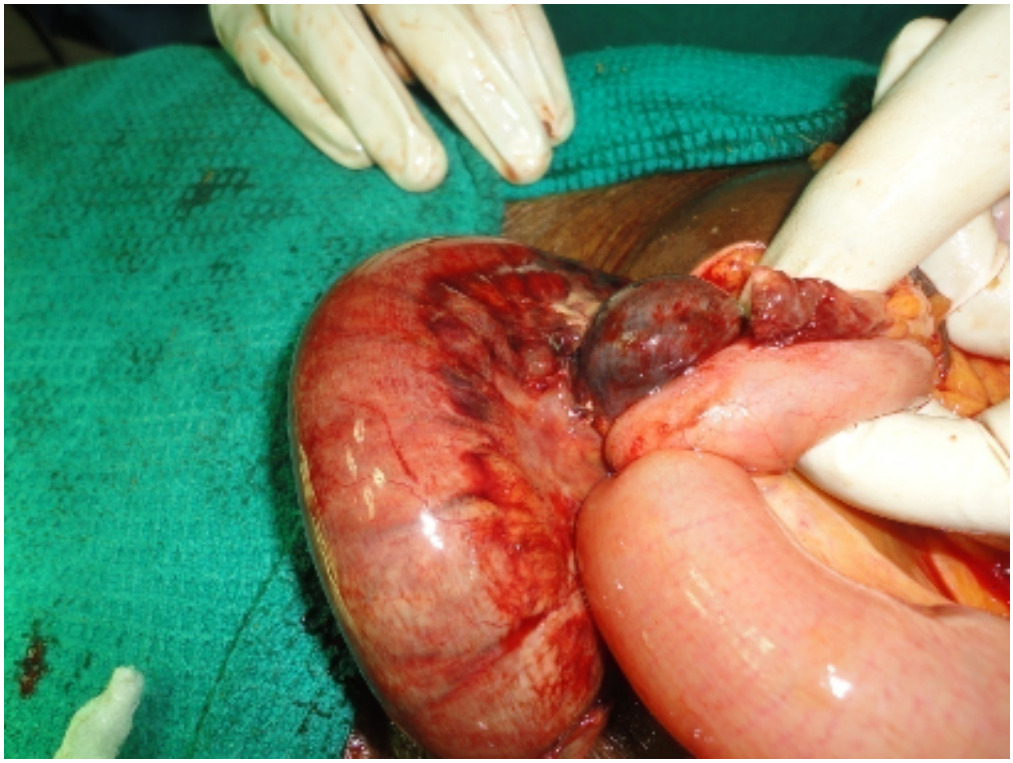
SBV presents with the classical features of intestinal obstruction. The outstanding symptom is central abdominal pain, the severity of which may be out of proportion to the apparent degree of obstruction. The diagnosis should be particularly considered if the pain does not respond to narcotic analgesia, although in such cases frank gangrene is often already present. Associated with vomiting, distension, tenderness, peritonism, absent bowel sounds, elevated temperature, tachycardia, and leucocytosis.

RADIOLOGICAL INVESTIGATIONS

On barium studies, the small bowel loops may show a typical ‘corkscrew’ or ‘spiral’ pattern suggesting SBV . On angiography, spiralling of the branches of the twisted superior mesenteric artery produces a "barber pole" appearance that suggests the diagnosis. On CT or MRI Characteristic findings include the ‘whirl’ sign of the rotated mesentery and ‘peacock’s tail’ sign (due to torsion of the bowel around the mesenteric axis) Small bowel ischaemia or infarction is suggested on CT by the presence of bowel wall thickening, intra-mucosal air and intra-peritoneal fluid.

Barium study – cock screw. CT abdomen-peacock’s tail





Intraoperative Picture of volvulus of small bowel with gangrene

TREATMENT

The surgical options for SBV consist of derotation, with or without fixation, and resection with anastomosis.. In view of the excellent blood supply of the small bowel, some authors recommend resection and primary anastomosis in all cases of SBV, regardless of whether gangrene is present or not.

POST OPERATIVE COMPLICATIONS

Major complications:

1. Anastomotic leak
2. Wound dehiscence
3. Septicaemia

4. Renal failure
5. Multiorgan failure

Minor complications:

1. Faecal fistula
2. Prolonged ileus
3. Wound infection
4. Intra abdominal abscess
5. Respiratory tract infection
6. Urinary tract infection
7. Deep vein thrombosis

Picture of faecal fistula at the drain site

Picture of wound infection

MATERIALS AND METHODS

All the acute abdominal cases admitted in surgical ward of Coimbatore medical college hospital during the period of October 2009 to October 2011 were studied. Out of all the admitted cases small bowel gangrene cases due to various etiology identified and 25 cases of them were included in our study.

INCLUSION CRITERIA

1. Patient presenting with acute abdomen and diagnosed as small bowel gangrene peroperatively.
2. Patients with strangulated hernia and gangrene of small bowel presenting with signs of peritonitis.

EXCLUSION CRITERIA

1. Patients less than 18 years and more than 80 years
2. Pregangrenous bowel which retained its viability intraopertively.

All the cases were thoroughly examined . Emphasis was given to the past history of any thromboembolic events , systemic hypertension, heart disease, and diabetes mellitus. Time of presentation after the onset of symptoms was noted . Patient presenting with acute abdomen and irreducible mass abdomen who were later diagnosed as small bowel gangrene per operatively were included in the study.

Investigations like complete hemogram, blood urea, serum creatinine, blood sugar, chest x-ray , x ray abdomen erect , ECG, blood grouping and typing, were done for all the patients pre operatively. Nasogastric tube was inserted in all cases and the amount and type of fluid aspirated was noted. All patients catheterized with foley's

catheter and urine output noted. 4 quadrant aspiration was done in all cases and aspirate was noted. Per rectal examination was done in all cases.

All the patients were resuscitated with iv fluid, broad spectrum antibiotics. Patients were taken up for surgery. Groin exploration was done for strangulated inguinal hernia, midline laparotomy for other patients presenting with abdominal pain suggestive of peritonitis ,obstructed ventral hernia and epigastric hernia patients.

Intra operative finding were noted . Gangrenous bowel were resected and continuity re-established by anastamosis of bowel. Thorough abdominal wash was given with normal saline. Repair of the hernia defect done in strangulated hernia cases. Abdomen is closed in layers after keeping flank drain.

Post operatively broad spectrum antibiotics given. Daily dressing of the wound changed. Daily drain output noted. Patients were kept nil per oral and sips of fluid started once the motility of the bowel regained. Wound infection cases were treated according to the pus culture and sensitivity report. Secondary suturing was done for the patients who developed wound gaping and burst abdomen. Relaparotomy was done for cases who developed anastomotic leak and the findings were noted. Patients developing fecal fistula were treated conservatively and daily output of the fistula is noted by applying colostomy bag. Parenteral nutritional support was given for patients who had prolonged ileus and for patients who developed fecal fistula. Patients who survived were discharged when the general condition improved and regular follow was done .

A proforma which included the history, clinical findings, investigations done, per operative finding, surgical procedure done, post operative complications was prepared for this study. Analysis of all the data was done and the common presentation, etiology, management and the outcome of the management were discussed in detail.

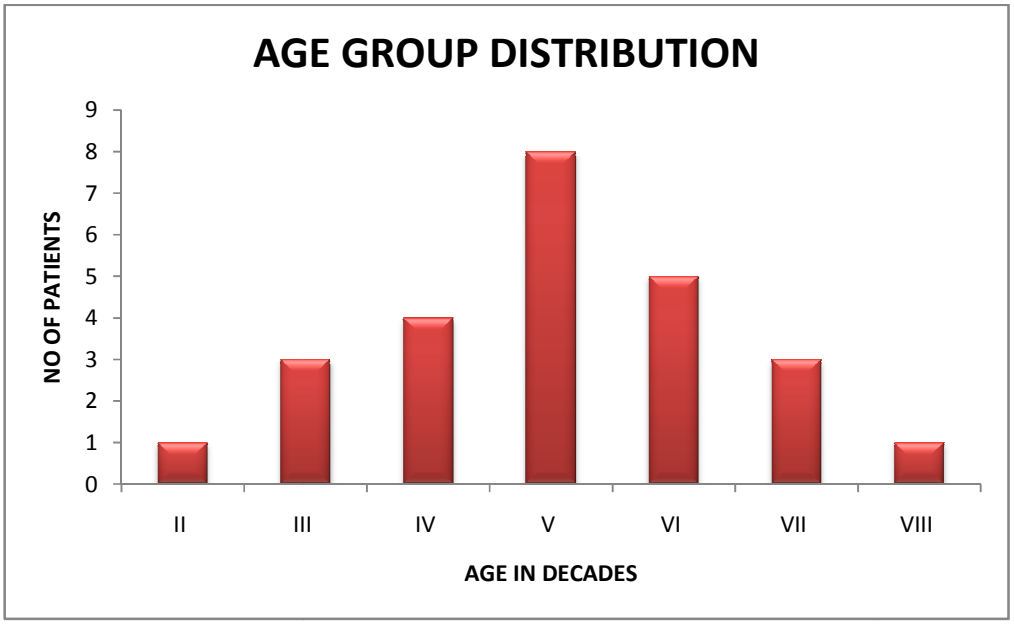
OBSERVATION AND RESULTS

AGE INCIDENCE

The age incidence in this study was between 19 years to 76 years.

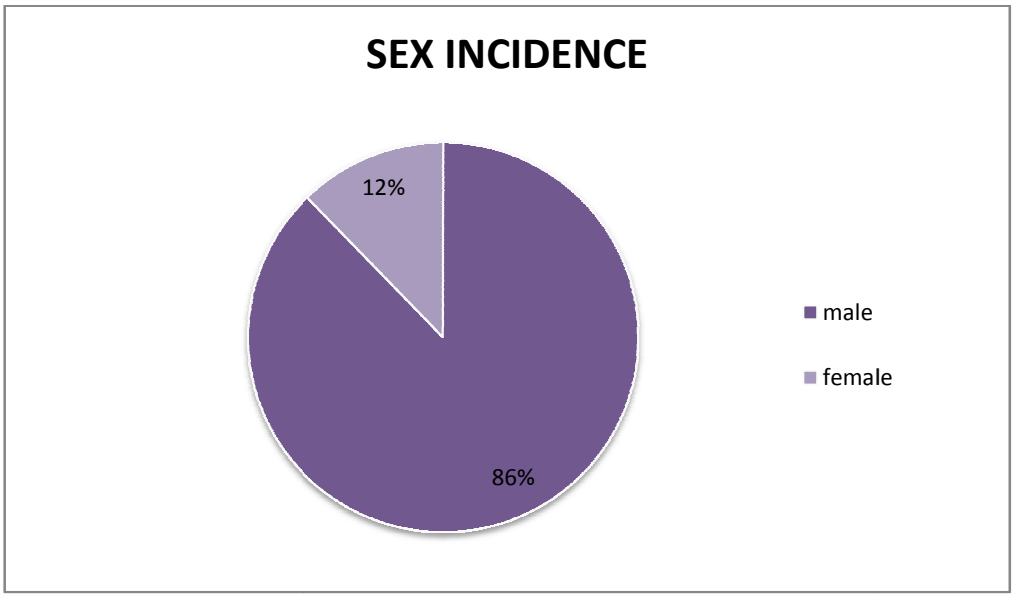
AGE GROUP DISTRIBUTION

AGE	NO OF PATIENTS	PERCENTAGE
10-20	1	4
21-30	3	12
31-40	4	16
41-50	8	32
51-60	5	20
61-70	3	12
71-80	1	4
TOTAL	**25**	**100%**



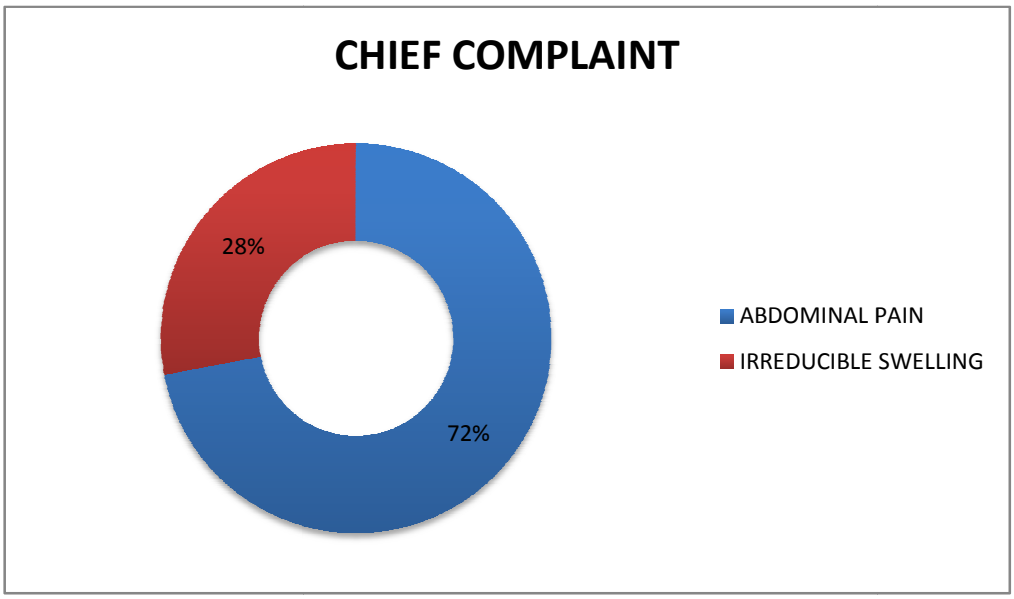
SEX INCIDENCE

Males are more predominantly affected with male 86% and female 12%



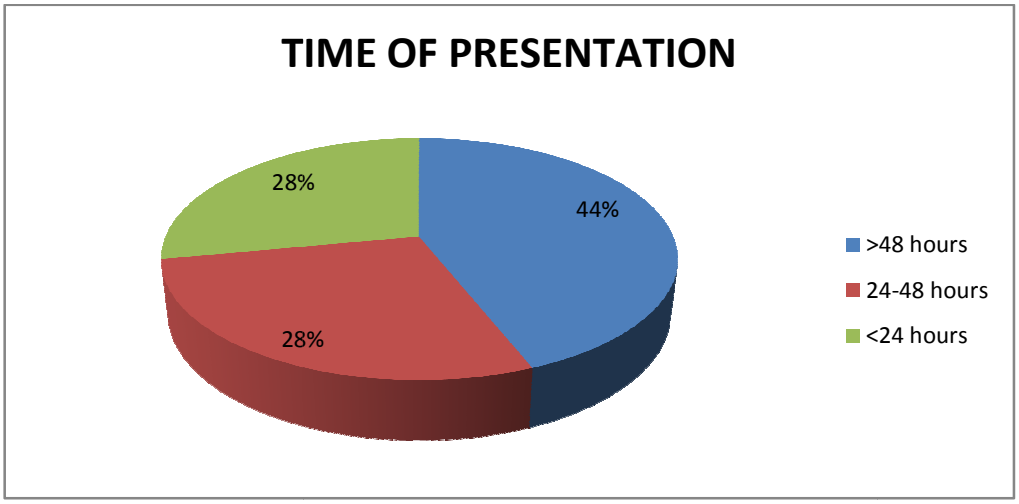
CHIEF COMPLAINTS

Most commonly presented with abdomen pain 72% and irreducible swelling 28%



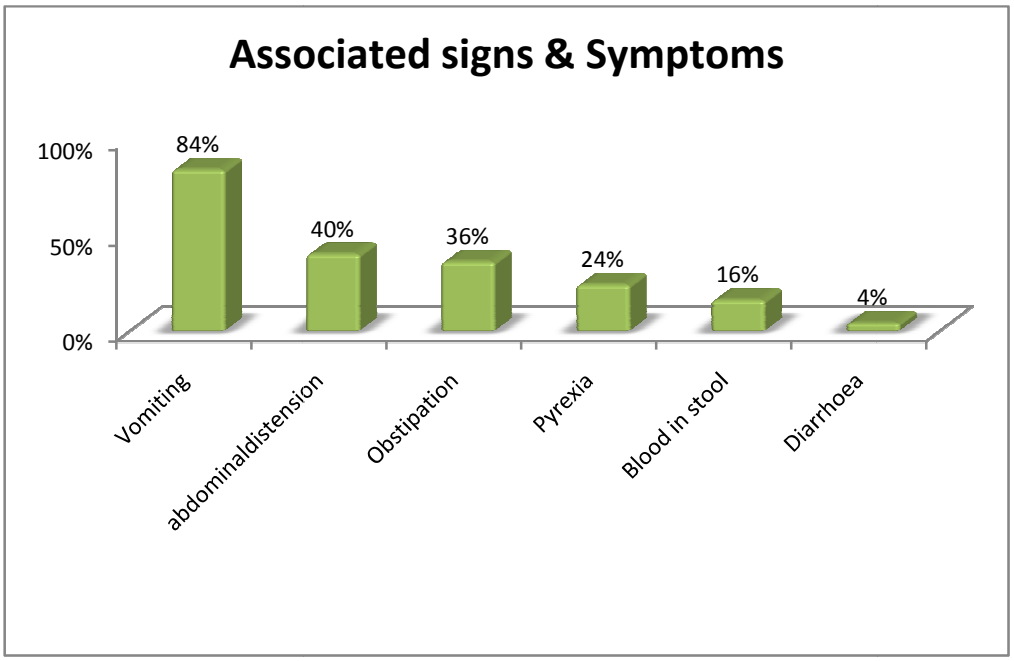
DURATION OF SYMPTOMS

44% of patients presented to the hospital >48 hours of onset of symptoms, 28% in 24-48 hours and 28% in <24 hours



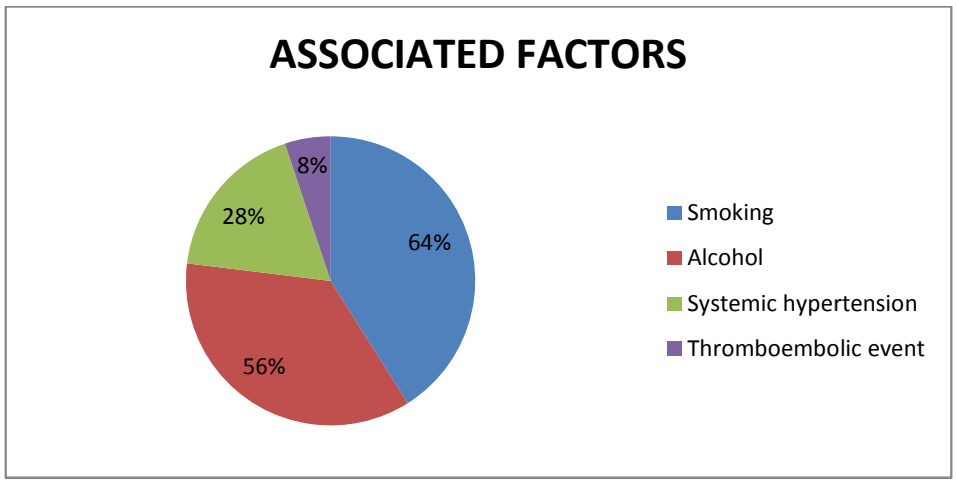
ASSOCIATED SYMPTOMS AND SIGNS

Most commonly abdominal pain associated with vomiting 84%, followed by abdominal distension 40%,obstipation 36%,pyrexia 24%,blood in stools 16%, diarrhoea 4%.

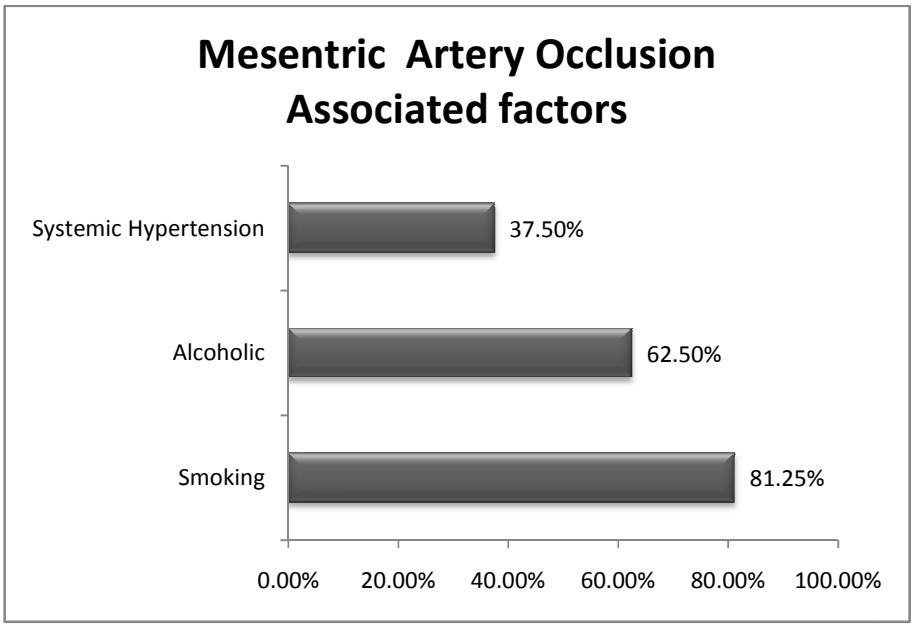


ASSOCIATED FACTORS

Most commonly associated factor out of 25 cases is Smoking- 64% Alcohol-14%, Systemic Hypertension-28%, Thromboembolic event-8%,



Out of 16 mesenteric artery occlusion cases 81.25% had history of Smoking,62.5% were Alcoholics, 37.5% were having Systemic Hypertension



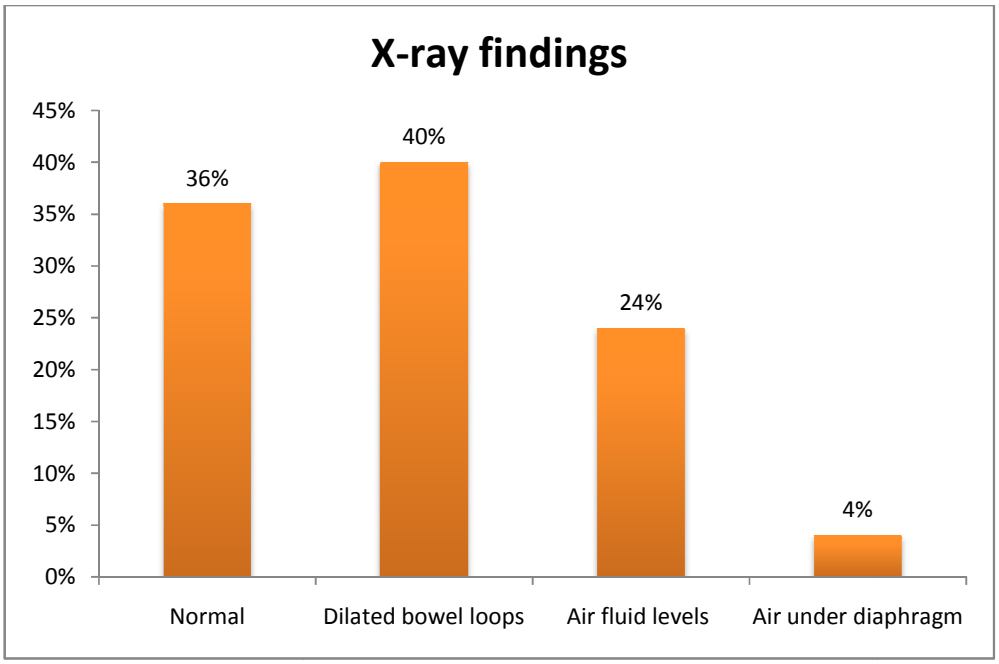
One case has previous history of laparotomy for blunt injury abdomen presented with constriction band .one patient had undergone bilateral hernioraphy presented with obstructed inguinal hernia.

PER RECTAL EXAMINATION

65%- Normal faecal staining, 24%- Blood stained faeces, 8%- Empty

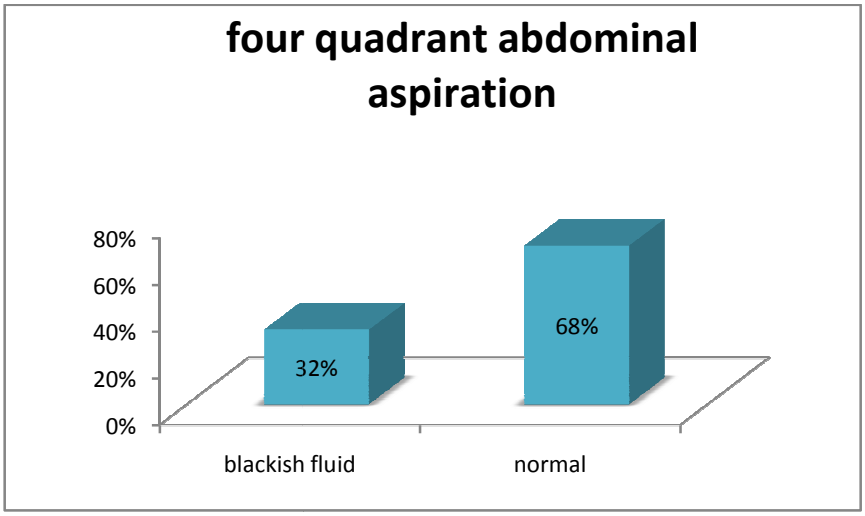
X-RAY ABDOMEN

Done in all patients. 36% - no significant finding, 40% - dilated bowel loops, 24% - air fluid levels, 4% - air under diaphragm



FOUR QUADRANT ABDOMINAL ASPIRATION

8 out of 25 cases showed blackish fluid aspiration



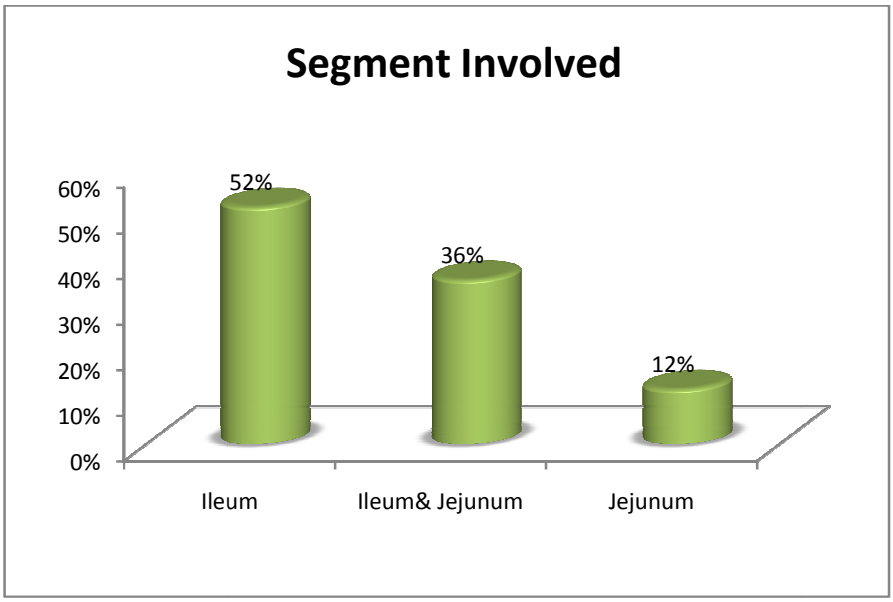
Out of these 8 cases 7 were mesenteric vascular occlusions and 1 was Ileal volvulus. All the patients were resuscitated with intravenous fluid, one dose of broad spectrum antibiotics along with inj.metronidazole.

2 patients came with circulatory shock and successfully resuscitated and taken up for surgery.

40% cases had leucocytosis and 12% had elevated renal parameters. Under general anaesthesia /regional anaesthesia laparotomy or inguinal exploration done .

SEGMENT OF BOWEL INVOLVED

On laparotomy gangrene segment identified as Ileum- 52%, Ileum & Jejunum-36%, Jejunum-12%

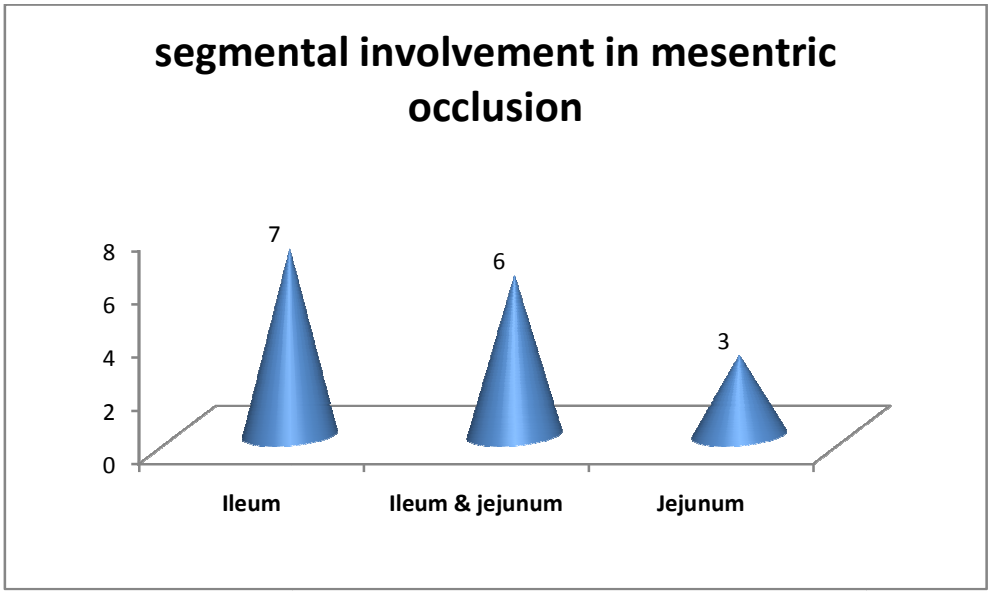


Out of 16 cases of mesenteric vascular occlusion,

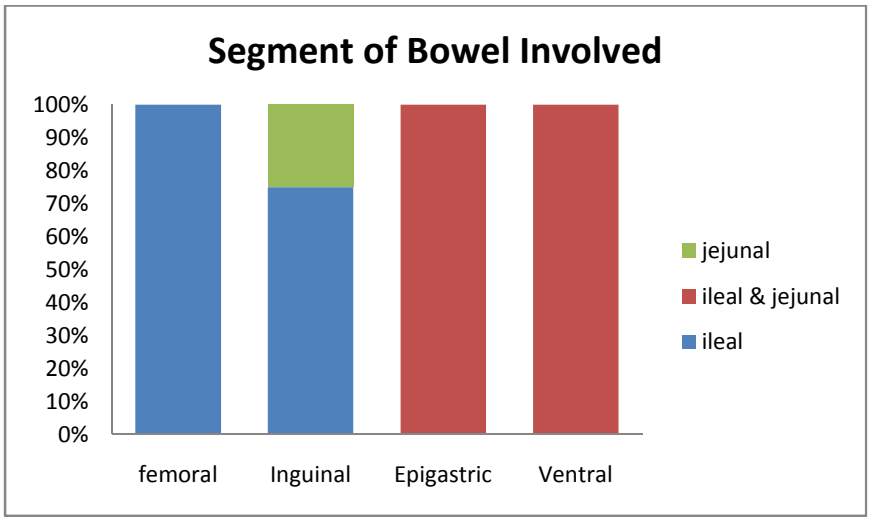
3- jejunum

7- Ileum

6- jejunum +ileum



Out of 7 Obstructed Hernia, 1 femoral was Ileal, 3 inguinal were Ileal, 1 inguinal was jejunal, 1 epigastric was Ileal&Jejunal, 1 ventral was ileal&jejuna. Out of the 3 inguinal hernia one was Richter's hernia involving ileum.

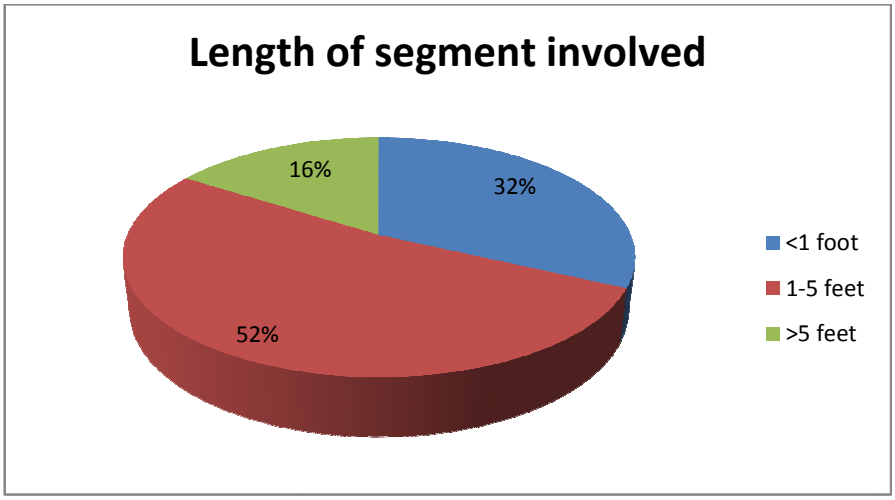


LENGTH OF SEGMENT INVOLVED

<1Foot – 32%,

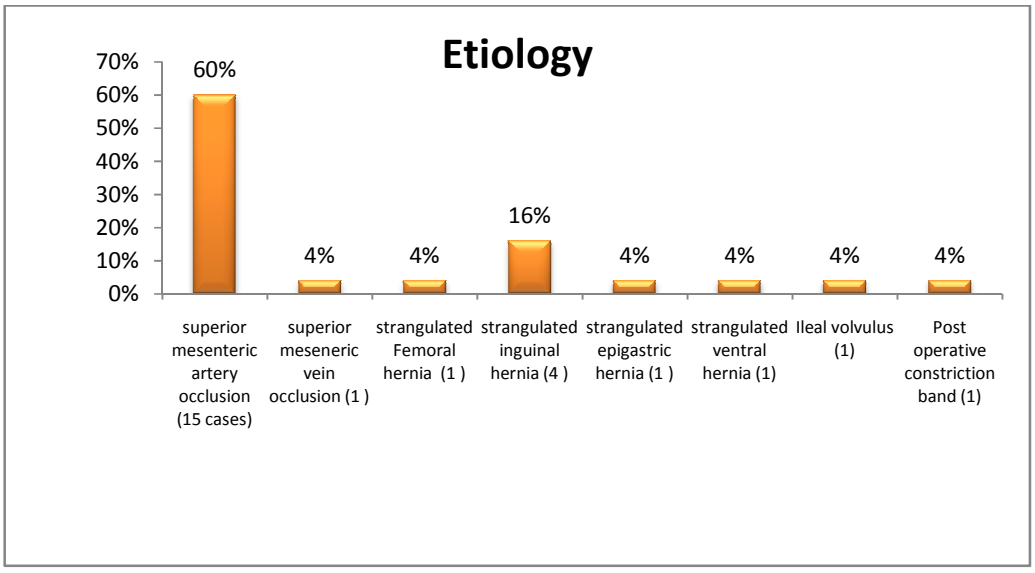
1-5 feet- 52%

>5 feet- 16%



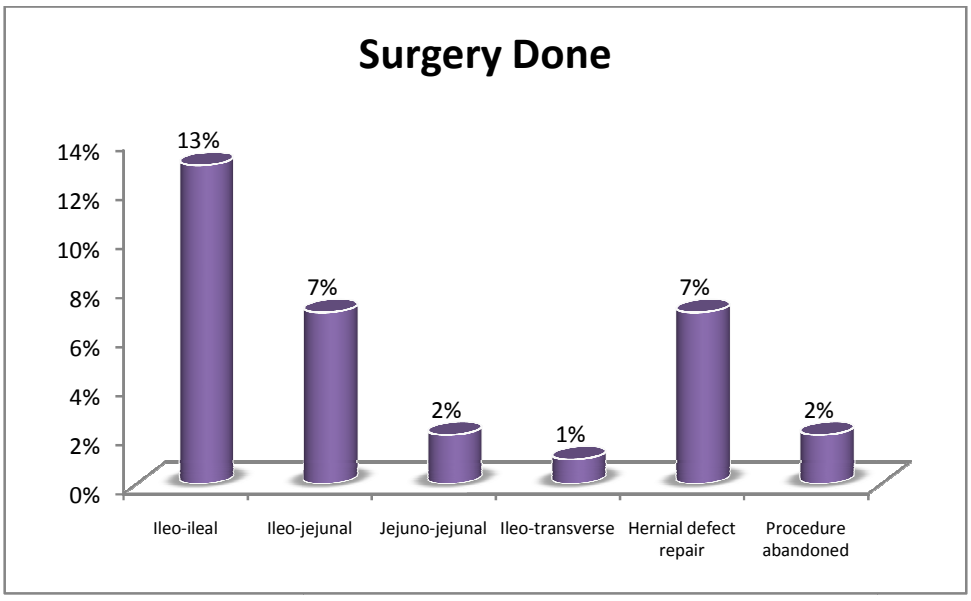
ETIOLOGY

60% had superior mesenteric artery occlusion, 4% had superior mesenteric vein occlusion, 4% femoral hernia, 16% Inguinal Hernia, 4% Epigastric Hernia, 4% ventral Hernia.



SURGERY

After resection of gangrenous bowel Ileo- ileal anastomosis- 13, Ileo-jejunal anastomosis- 7, Jejuno-jejunal anastomosis- 2, Ileo- transverse anastomosis- 1, Hernia defect repair- 7, procedure abandoned- 2 cases because of entire small bowel gangrene.



POST OPERATIVE FOLLOW UP

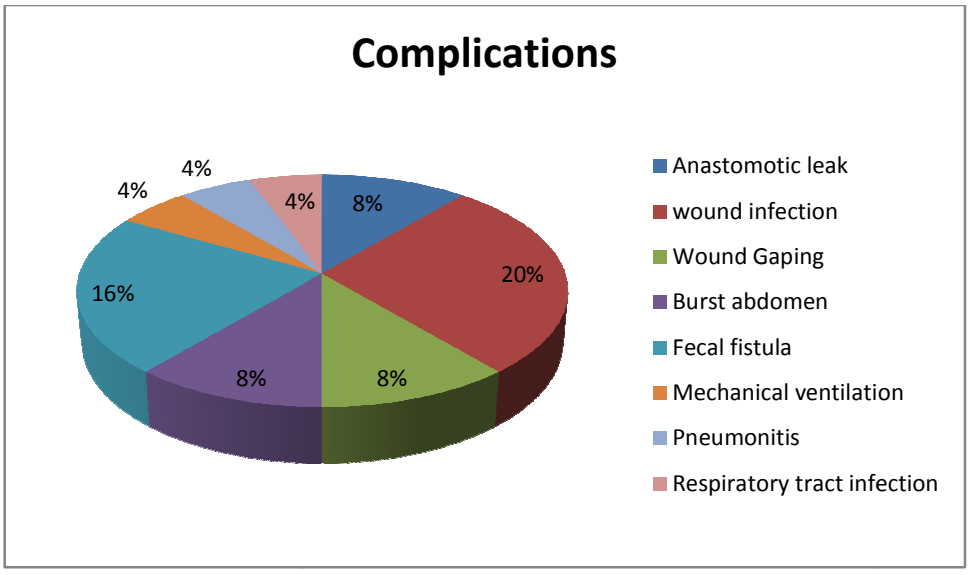
All the patient was given broad spectrum antibiotics and inj.metronidazole for 7 days. Five patients developing wound infection and was treated according to the wound culture and sensitivity. Daily examination of laparotomy wound and drain site done. All patients catheterized until adequate output maintained and continuous RTA aspiration done until <20ml aspirated for 24hours.

One patient required mechanical ventilator support and extrubated on 1st POD. The patients with systemic hypertension managed accordingly.

One patient with mitral stenosis and atrial fibrillation and developed acute left lower limb ischemia due to embolus in common femoral artery on 3rd POD day. Immediate embolectomy done.

COMPLICATIONS

Anastomotic Leak-8%, Wound infection- 20%, Wound gaping-8%, Burst abdomen-8%, Faecal fistula-16%, Pneumonitis-4%, Respiratory tract infection-4%, mechanical ventilation-4%



2 patients developed anastomotic leak and relaparotomy was done and reanastomosis done . Both of them died.

5 patients developed wound infection . pus culture and sensitivity was done and treated accordingly. 2 patients developed wound gaping. Secondary suturing was done .

2 patients had burst abdomen. Emergency tension suturing was done. Both the patients died.

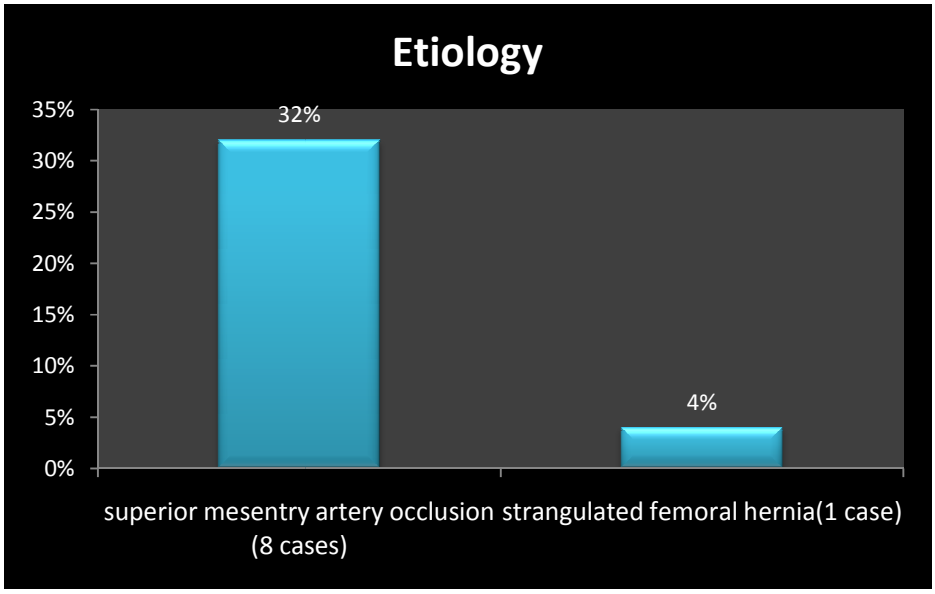
1 patient required post operative ventilator support and weaned off successfully on 1st POD.

4 patient developed faecal fistula. 3 cases from drain site and 1 from laparotomy wound. All were treated conservatively. 2 patients improved well and 2 died.

1 patient developed pneumonia and one developed respiratory tract infection. Sputum culture and sensitivity was done and treated accordingly. Chest physiotherapy and bronchodialators was given. Both of them recovered well .

MORTALITY

In this study mortality was found in 9 cases. 8 cases of superior mesenteric artery occlusion and 1 case of strangulated femoral hernia.



MORBIDITY AND MORTALITY ACCORDING TO LENGTH OF BOWEL RESECTED

LENGTH	<1 FEET	1-5 FEET	>5 FEET
MORTALITY	4% (1)	20% (5)	12% (3)
MORBIDITY	12%(3)	36% (9)	4% (1)

MORTALITY AND MORBIDITY ACCORDING TO THE TIME OF PRESENATION

DURATION	<24 HOURS (7)	24-48 HOURS (7)	>48 HOURS (11)
MORTALITY	8%(2)	12%(3)	16%(4)
MORBIDITY	12%(3)	8%(2)	36%(9)

PROBABLE CAUSE OF MORTALITY IN 9 CASES

1. 2 patients presented with full length of small bowel gangrene developed septicaemia with multi organ failure.
2. 1 patient developed high output fistula with septicaemia
3. 2 patient developed anastomotic leak and relaparotomy was done ; developed septicaemia shock
4. 1 patient of HOCM with atrial fibrillation
5. 1 patient developed burst abdomen and septicaemia shock
6. 2 patients had hypotensive shock.

DISCUSSION

Small bowel gangrene recognized as potential cause of mortality. It occurs due to various causes. This study was under taken to study the ways of presentation and analyze the methods to improve the outcome of small bowel gangrene.

The youngest patient is 19 years old presented with post operative constriction band causing gangrene of ileum. The oldest is 76 years resented with strangulated Richter's hernia. The peak incidence is in 5^{th} decade with 32%. The most common cause was mesenteric artery occlusion. The mortality rate was found to be high in 5^{th} decade with 12%(3) due to mesenteric vascular occlusion, 1 patient had hypertrophic obstructive cardiomyopathy with atrial fibrillation and 1 patient presented with entire small bowel gangrene. Above 70 years of age strangulated hernia was common.

Bowel gangrene is more common in males 86% when compared with females 12%. Strangulated hernia commonly occur in male due to increased physical activities and strenuous effort. Mesenteric vessel occlusion is also common in males may be due to associated thrombogenic factors like smoking and alcohol.

The most common complaint of presentation was abdominal pain 72%. The pain was started at one quadrant initially and later spread all over the abdomen indicating the progression of the bowel infraction.

25% cases presented with irreducible swelling in the abdomen as strangulated hernia; 16% presented as inguinal,4% as femoral, 4% as epigastric,4% as ventral hernia.

The most common associated symptom is vomiting 84% due to obstruction and paralytic ileus from strangulated hernia. 40% cases presented with abdominal distension due to accumulation of toxic fluid and gaseous distension due to onset of gangrene. Obstipation was seen in 36% of cases due to strangulated hernia. 24% cases had fever indicated the onset of tissue necrosis and peritonitis.

The most common associated factor was smoking 64% followed by alcoholism 56% and systemic hypertension 28%.out of 16 cases of mesenteric artery occlusion 13 patients were smokers, 10 patients alcoholic and 6 patients had systemic hypertension. This indicates that the occlusion of mesenteric vessel was higher in patient with history of smoking and alcoholic.

Out of 25 patients only 4(16%) patients presented with blood in stool, 6(24%) patients with blood stained feces on per rectal examination. This find was not present in all cases and percentage was also low. Hence this finding is not useful in arriving at the diagnosis.

X-ray abdomen in erect position was taken in all patients. 24% showed air fluid level most because of obstruction due to strangulated hernia. Most of the x-ray findings were inconclusive of gangrene. One case had air under diaphragm due to perforation of the gangrenous bowel.

Only 2 patients presented with features of shock. 1 patient had entire small bowel gangrene and 1 patient had superior mesenteric vein occlusion with 1.5 feet of ileal gangrene. Both the patients were resuscitated and taken up for surgery. Entire bowel gangrene patient died post operatively and the other patient recovered well.

Four quadrant aspiration was done in all patients and toxic haemorrhagic fluid aspirated in 32%(8)cases. Out of 8 cases 7 cases were mesenteric vessel occlusion and 1 was ileal volvulus. So paracentesis can be relied on diagnosis of bowel gangrene that is not due to strangulated hernia as all the strangulated hernia patients showed negative aspiration.

In this study most common etiology of small bowel gangrene superior mesenteric artery occlusion 60%(15). 4 cases had predisposing factor for embolus formation such as mitral stenosis with atrial fibrillation, hypertrophy cardiomyopathy with atrial fibrillation, liver failure, past H/O of cerebro vascular accident ,coronary artery disease. Other cases had predisposing factors for atherogenic thrombus occlusion such as smoking, alcoholic, systemic hypertension, above of 50 years(5)

Strangulated inguinal hernia seen in 16%(4), 4%(1) presented with strangulated Richter's hernia. Strangulated femoral hernia 4%, strangulated ventral hernia4%,strangulated epigastric hernia 4%.

Ileal volvulus 4%(1).

Post operative constrictive band 1%.

Gangrenous segment of the bowel is resected and end to end anastomosis done depending upon which segment was resected. In all cases except one in which ileotransverse side to side anastomosis done because of extensive involvement of ileum upto ileocaecal junction. All the anastomosis were closed in 2 layers.

More the length of the bowel resected add to increased risk of mortality due to prolonged time of surgery in already haemodynamicaly compromised patients and also results in increased post operative complication. 1-5 feet of bowel resection lead

to 20%(5) mortality, 36%(9) morbidity. 3 patient had >5 feet resected all of them died. <1 foot resection resulted in 4% mortality and 12% morbidity. 2 cases presented as entire small bowel gangrene as found per operatively and no furthers surgical resection done. Both of them died post operatively.

The delayed time of presentation shown to influence the mortality and morbidity. In this study mortality is high in patient presenting >48 hours late after onset of symptoms(16%)and 12% for 24-48 hours delayed presentation and 8% for <24 hours delay. Out of 8 cases of mortality due to artery occlusion 4 cases presented > 48hours (50%). This is due to prolonged exposure of bacterial toxin in the peritoneal cavity leading to onset of septicaemia. In a report from Madrid of 21 patients with SMA embolus, intestinal viability was achieved in 100% if the duration was <12 hours. 56% for 12-24 hours. 18% for >24hours.

Increased morbidity was seen in patients with >48 hours delay (36) such as wound infection, wound gaping, RTI, faecal fistula, post operative ventilator support for reduced respiratory effort, burst abdomen. 2 cases required relaparotomy for correction of anastomotic leak. In <24 hours lesser complication like wound infection was seen.

Seven patients presented with strangulated hernia out of which one died that is 14%. When compared with over all mortality of small bowel gangrene it is 4% in this study.

In this study mortality can be considered in two aspects as mortality due to mesenteric vessel occlusion and mortality due to strangulated hernia.

MORTALITY DUE TO MESENTERIC VESSEL OCCLUSION	
AUTHOR	**MORTALITY PERCENTAGE**
Woosup M Park USA	63%
Joaquine Marchena Spain	64.5%
In this study	50%

MORATLITY DUE TO STRANGULATED HERNIA	
AUTHOR	**MORTALITY**
M-Ohene Yeboah Kumasi	11.8%
William O.Barnet Mississipi	20%
In this study	14%

When compared with other international studies the mortality percentage difference may be due to less number of cases include in our study .

Considering the high mortality rate of patients with intestinal gangrene, prevention seems to be most logical statergy to improve outcome if the high risk group can be identified. High risk group include patients with

1. Past H/O of thromboembolic events.
2. Rheumatic heart disease.
3. Arrhythmias.
4. Patients taking digitalis, diuretics, oestrogen, danazol.

5. Patients with protein C& protein S deficiency, anti thrombin III deficiency.

6. Sickle cell disease.

In this case study 2 patients (8%) presented with atrial fibrillation(AF) , 1 patient with past H/O of cerebro vascular accident(CVA), coronary artery heart disease(CAHD), aortic stenosis. Treatment of chronic AF with anticoagulant or cardioversion and treatment of CVA, CAHD has been demonstrated to significantly reduce thromboembolic complication. In this study ,previous proper treatment of patient with AF would have prevented the mortality due to embolic bowel gangrene and morbidity due to embolectomy. Four patients (16%) presented with the co morbid condition and 1 patient died (4%).

Patient should be educated regarding the benefits of regular treatment and complication of stopping the drug without physician's advice. High risk patients should be advised to seek immediate medical attention if they develop abdominal pain to rule out thrombo embolic occlusion earlier. Physician at the primary health care level should be educated about the possibility of mesenteric vessel occlusion in high risk patient and refer them to higher centre immediately.

Diagnosis of bowel gangrene can made if patient present s with acute abdomen pain, tachycardia, fever, leucocytosis, abdomen tenderness, rigidity with history of smoking, hypertension, past thrombo embolic event, RHD,AF and abdominal paracentesis showing hemorrhagic fluid.

All hernia should be operated as soon as diagnosed to prevent obstructed and strangulated hernia. In this case study early repair of hernia would have prevented 4% mortality from strangulated hernia and 12% of morbidity due to wound gaping ,wound infection and respiratory infection.

After the patient reaching the hospital the following should be done to reduce the mortality and morbidity rate of bowel gangrene :

1. Early antibiotic therapy – this will post pone the irreversible stage of septicaemia. They protect against the emerging bacteraemia.

2. No unnecessary delay in surgery if peritonitis of unknown etiology suspected because this will lead to irreversible hypotension due to prolonged exposure of bacterial toxins.

3. Judicious decision should be made between early surgical management to terminate exposure to gangrenous bowel toxin versus the adequate pre operative time in maximally correcting fluid and electrolyte imbalance.

4. Proper and correct technique of resection anastomosis of the bowel to be followed and the gangrenous part should be resected out immediately. The operative time should be reduced as much as possible.

5. Second look laparotomy wil be helpful when the survival of the bowel could not be assessed in emergency laparotomy and in patients with unresolved signs of peritonitis to rule out the progression of gangrene.

CONCLUSION

1. Highest incidence seen in 5th decade.
2. Males are more predominantly affected than females.
3. Superior mesenteric vessel occlusion and strangulated abdominal hernias are common cause of small bowel gangrene.
4. Patient presented with chief complaints of abdominal pain and irreducible swelling over the inguino scrotal region. But clinical parameters are less reliable in diagnosis. Suspicion of gangrene can be made when patient present with abdominal pain associated with smoking, systemic hypertension, past H/O of thrombo embolic event, RHD,AF, abdominal guarding rigidity ,fever and tachycardia.
5. Most common factor associated with mesenteric vessel occlusion is smoking and alcoholic.
6. Preoperative aspiration showed haemorrhagic fluid in 44%cases of bowel gangrene caused by other than strangulated hernia.
7. Mortality rate is directly proportional to the length of the bowel involved .
8. Delayed hospitalization after the onset of symptoms significantly increase the morbidity and mortality.
9. From this study it is inferred that the delay in hospitalization, length of the bowel involved influence the morbidity and mortality.
10. Early diagnosis and early intervention is the key to reduce the mortality rate.

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PROFORMA

I. Name: **I.P.No:** **Case no:**

Age: Date of symptom onset:

Date of admission:

Sex: Date of operation:

Religion: Date of discharge:

Address:

II. **PRESENTING COMPLAINTS:**

1.

2.

3.

4.

III. **DETAILED HISTORY OF PRESENTING ILLNESS:**

1. PAIN:
    a. Time and mode of onset:
    b. Situation:
    c. Character: type at onset/type at present

        Colicky /burning /throbbing/

        Sever agonizing pain.
    d. Progress:
    e. Radiation:
    f. Related to bowel and micturition:
    g. Relation to food:

2. VOMITING:
    a. Present /absent.
    b. Character: Projectile / regurgitation.
    c. Frequency.
    d. Quality. – Nature
        - Colour
        - Taste
        - Odour.
    e. Quantity
    f. Associated material with vomitus.
    g. Duration - continuous/ intermittent.
    h. Progress.
    i. Relation to pain:
    j. Haematemesis: Present /absent
        Colour
        Frequency
        Amount
3. DISTENSION OF ABDOMEN:
    a. Mode of onset: gradual /sudden.
    b. Site
    c. Duration
    d. Progress
4. BOWELS:
    a. Tenesmus
    b. Constipation

c. Diarrhoea

d. Dysentery

e. Melena

5. FEVER: since days

Continuous / intermittent/ remittent.

6. MICTURATION.

IV. **PAST HISTORY:**

a. Previous history of similar attacks:

b. Any previous abdominal surgery:

c. Hypertension

d. Tuberculosis

e. Diabetes

f. Jaundice

g. Drug allergy

V. FAMILY HISTORY:

a. Members – any member of family suffered from similar attacks.

b. Health status

VI. **PERSONAL HISTORY:**

a. Diet: vegetarian/mixed.

b. Habits: smoking yes/no

Alcohol yes/no

Betel nut yes/no

c. Bowel/bladder habits:

d. Sleep:

e. Appetite:

VII. MENSTURAL /OBSTETRIC HISTORY IN FEMALES:

VIII. SOCIO-ECONOMIC HISTORY

IX. **GENERAL EXAMINATION:**

a. Build

b. Nourishment:

c. Eyes:

d. Mouth:

e. Nails:

f. Cyanosis:

g. Pedal oedema:

h. Lymphadenopathy:

i. Pulse:

j. B.P. mm of hg :

k. Respiration:

l. temperature:

m. jaundice

n. dehydration

X. LOCAL EXAMINATION:

1. Abdomen:
    a. Inspection :
        1. Shape:

2. Movements:
3. Umbilicus:
4. Distension: local/general.
5. Visible peristalsis:
6. Visible mass – site
   -size
   -shape
   -surface
   -borders
   -impulse on coughing.
7. renal angle: fullness:+/-
8. hernia sites:

b. Palpation:

1. local rise of temperature
2. tenderness site
3. swelling: situation
   size
   shape
   extent
   surface
   consistency
   movement

C. PERCUSSION:

D. AUSCULTATION:

XI. EXTERNAL GENITALIA:

XII. P/R EXAMINATION AND P/V EXAMINATION IN FEMALES:

XIII. SYSTEMIC EXAMINATION:

a. Respiration system

b. Cardiovascular system:

c. Central nervous system.

XIV. PROVISIONAL DIAGNOSIS:

XV. FINDINGS FOR DIAGNOSIS

XVI. INVESTIGATIONS

1. BLOOD INVESTIGATION:
   a. Hb%
   b. Blood grouping
   c. TC,DC,ESR
   d. Blood urea, serum creatinine
   e. Random blood sugar
   f. Serum electrolytes
2. URINE EXAMINATION
   a. Albumin
   b. Sugar
   c. Microscopy
3. RADIOLOGICAL INVESTIGATIONS
   a. Plain x ray of abdomen
   b. Contrast studies
   c. Other relevant radiological investigations

4. Ultrasound examination
5. CT scan
6. M.R.I
7. Arteriography
8. Laproscopy
9. 4 quadrant aspiration
10. stool examination

XVII. FINAL DIAGNOSIS

XVIII. TREATMENT

1. Treatment during resuscitation
2. Operation
3. Operative findings
4. Preoperative diagnosis
5. Post operative diagnosis
6. Post operative treatment
7. Post operative complication
8. Advice on discharge
9. Conclusion
10. Biopsy report

XIX. FOLLOW UP

1ST Follow up(1st week)

2nd follow up(1 month)

3rd follow up(3months)

MASTER CHART

SI NO	NAME	AGE	SEX	IP NO	ABDOMEN PAIN	IRREDUCIBLE SWELLING	HISTORY						SYMPTOMS					SIGNS					
							DURATION	DIABETIC	SHT	SMOKER	ALCOHOLIC	TEE	VOMITTING	DIAHHOREA	BLOOD IN STOOL	OBSTIPATION	PYREXIA	ABD DISTENSION	GUARDING/RIGIDITY	TENDERNESS	BOWEL SOUNDS	PER RECTAL	SHOCK
1	RAJESH	30	M	29438	+	-	>48	-	-	+	+	-	+	-	-	+	-	-	-	+	_	N	-
2	PALANISAMY	55	M	30246	+	-	>48	-	-	+	+	-	+	-	-	-	-	+	+	+	-	N	-
3	ANNADURAI	40	M	30254	+	-	>48	-	-	+	-	-	-	-	-	-	-	+	+	+	-	N	-
4	DHANASEKARAN	55	M	33249	-	IS	24	-	-	-	+	-	+	-	-	+	-	-	-	-	+	E	-
5	ELANGOVAN	35	M	50568	+	-	>48	-	+	+	-	-	+	-	-	-	+	+	+	+	+	N	-
6	NAVANETA KRISNAN	42	M	50113	-	IS	>48	-	-	+	+	-	-	-	-	-	+	-	-	-	-	E	-
7	SEMALAIAPPAN	50	M	53678	+	-	>48	-	+	+	+	-	+	-	+	+	+	+	+	+	-	BS	-
8	RAJU	35	M	65647	+	-	24	-	-	+	+	-	+	-		+	-	-	+	+	-	N	-
9	ABDUL JAFFER	64	M	12054	+	-	24	-	+	+	-	-	+	+	-	-	-	-	+	+	-	N	-
10	BABU	45	M	11496	+	-	>48	-	-	+	+	+	+	-	+	-	-	-	+	+	-	BS	+
11	KALIDAS	31	M	23843	+	-	48	-	-	+	-	-	+	-	+	-	-	+	+	+	-	BS	+

12	PALRAJ	19	M	32007	+	-	24	-	-	-	-	-	+	-	-	-	-	-	+	+	-	N	-
13	ARYAMMAL	70	F	35681	+	-	48	-	-	-	-	-	+	-	-	-	-	+	+	+	-	N	-
14	THANGAVEL	52	M	43634	-	ES	24	-	+	+	+	-	-	-	-	+	-	-	-	+	+	N	-
15	KITTUSAMY	55	M	51263	+	-	48	-	-	+	+	-	+	-	-	-	+	-	+	+	-	N	-
16	RAMAN KUMAR	24	M	53884	-	IS	48	-	-	-	-	-	+	-	-	-	-	-	-	+	+	N	-
17	RANGASAMY	45	M	28917	+	-	48	-	+	+	+	-	+	-	-	-	-	+	+	+	+	N	-
18	JANAGI	60	F	12303	-	SU	>48	-	-	-	-	-	+	-	-	+	-	-	-	+	-	N	-
19	MURUGAN	46	M	44555	+	-	>48	-	-	-	+	-	+	-	-	-	-	+	+	+	-	BS	-
20	SURESH	22	M	61394	+	-	>48	-	-	+	+	-	+	-	-	-	+	-	+	+	+	N	-
21	VENKATESAN	76	M	58396	-	IS	>48	-	-	-	-	-	+	-	-	-	-	-	-	-	+	N	-
22	MASILAMANI	45	F	55332	+	-	24	-	+	-	-	+	+	-	+	-	-	+	+	+	-	BS	-
23	KUSALAVAN	47	M	54506	+	-	48	-	+	+	+	-	-	-	-	+	+	+	+	+	-	BS	-
24	RAJAPPAN	50	M	54405	+	-	48	-	-	-	-	-	+	-	-	+	-	-	+	+	-	N	-
25	MURUGAN	63	M	45791	-	IS	24	-	-	+	+	-	+	-	-	+	-	-	+	+	-	N	-

SHT – SYSTEMIC HYPERTENSION, TEE – THROMBOEBOLIC EVENTS, IS – INGUINAL SWELLING, ES- EPIGASTRIC SWELLING, SU – SUB UMBILICAL , N- NORMAL, E- EMPTY, BS-BLOOD STAINED

INVESTIGATION							GANGRENE SEGMENT	LENGTH OF GANGRENE SEGMENT (FEET)	DIAGNOSIS	SURGERY	COMORBIDITY	MORBIDITY	MORTALITY
S.NO	LEUCOCYTOSIS	UREA/CREATININE	X RAY ABD	FOUR QUADRANT ASPIRATION	USG ABD	ECG							
1	-	N	NS	-	+	N	J	1	MAO	R & JJ A	-	AL,RL	+
2	-	N	NS	-	N	N	J	2.5	MAO	R& JJA	-	WI	-
3	-	N	DBL	-	+	N	J+I	1.5+0.5	MAO	R& JIA	-	WG	+
4	-	N	AFL+ AUD	-	+	N	I	0.3	SFH-L	R& IIA,HR	-	BA	+
5	+	N	DBL	-	+	N	I	0.5	MAO	R& IIA	-	-	-
6	-	N	DBL	-	+	N	I	0.5	SIH-L	R& IIA,HR	-	-	-
7	+	E	DBL	+	+	+	J+I	5	MAO	R& JIA	-	BA,AL,RL	+
8	+	N	NS	-	+	N	J+I	0.5+0.5	MAO	R& JIA	-	WI	-
9	-	N	DBL	-	+	+	J+I	6.5+10	MAO	R& JIA	HOCM,AF	-	+
10	+	E	DBL	+	+	+	I	1.5	MVO	R& IIA	CVA,CAHD,AS	VS	-
11	+	N	DBL	+	+	N	J+I	20	MAO	PA	-	-	+

12	-	N	NS	+	N	N	I	1	POCB	R & IIA	-	-	-
13	+	N	AFL	+	+	N	I	0.75	IV	R & IIA	-	-	-
14	-	N	NS	-	+	+	J+I	0.5	SEH	R & IJA,HR	LC,PHT, CCF,TBA	-	-
15	-	N	NS	-	N	N	I	1	MAO	R & IIA	-	-	+
16	-	N	AFL	-	+	N	I	1	SIH-R	R & IIA,HR	-	-	-
17	-	N	DBL	-	+	+	I	0.5	MAO	R & IIA	-	-	-
18	-	N	AFL	-	+	N	I+J	0.5	SVH	R & JIA,HR	-	WI	-
19	-	N	NS	+	+	N	I	3	MAO	R & IIA	-	FF	+
20	+	N	NS	-	+	N	I	2	MAO	R & IIA	-	FF	-
21	-	N	DBL	-	+	N	I	-	SRH	R & IIA,HR	-	WG,RI	-
22	+	E	DBL	-	+	+	J	3	MAO	R & JIA,EL	MS,AF, CFAB,LAC	-	-
23	+	N	AFL	+	+	+	I+J	20	MAO	PA	-	FF,WI	+
24	-	N	NS	+	+	N	I	3	MAO	R&IIA	-	FF,P	-
25	+	N	AFL	-	+	N	J+I	10	SIH-R	R& ITA	-	WI	-

ABD – ABDOMEN**, N** – NORMAL**, E** – ELEVATED, **NS** – NOT SIGNIFICANT, **DBL**- DIALATED BOWEL LOOPS, **AFL**- AIR FLUID LEVEL, **AUD**- AIR UNDER DIAPHRAGM**, I**- ILEUM**, J** – JEJUNUM, **MAO**- MESENTERIC ARETRY OCCLUSION, **MVO** – MESENTERIC VEIN OCCLUSION**, SFH-L**-STRANGULATED FEMORAL HERNIA LEFT, **SIH-L-**STRANGULATED INGUINAL HERNIA LEFT**, POCB**- POST OPERATIVE CONSTRICTIVE BAND, **IV**- ILEAL VOLVULUS**, SEH** – STRANGULATED EPIGASTRIC HERNIA, **SIH-R-**STRANGULAED INGUINAL HERNIA- RIGHT, **SVH** – STRANGULATED VENTRAL HERNIA, **SRH**- STRANGULATED RICHTER'S HERNIA**, R-** RESECTION**, JJA** – JEJUNOJEJUNAL ANASTOMOSIS**, JIA**- JEJUNOILEAL ANASTOMOSIS**, IIA** – ILEOILEAL ANASTOMOSIS, **HR-** HERNIORAPHY**, PA-** PROCEDURE ABDONDED, **EL** – EMBOLECTOMY, **ITA**- ILEOTRANSVERSE ANASTOMOSIS**, HOCM** – HYPERTROPHY OBSTRUCTIVE CARDIOMYOPATHY**, AF-** ATRIAL FIBRILATION, **CVA**-CEREBOVASCULAR ACCIDENT**, CAHD**- CORONARY ARTERY HEART DISEASE, **AS**- AORTIC STENOSIS**, LC**- LIVER CIRRHOSIS**, PHT** – PULMONARY HYPERTENSION**, CCF** –CONGESTIVE CARDIAC FAILURE**, MS-** MITRAL STENOSIS, **CFAB**- COMMON FEMORAL ARTERY BLOCK**, LAC**- LEFT ATRIAL CLOT**, AL** – ANASTOMOTIC LEAK, **RL-** RELAPROTOMY, **WI-** WOUND INFECTION, **WG**- WOUND GAPING**, BA-**BURST ABDOMEN**, VS-** VENTILATORY SUPPORT**, FF** – FAECAL FISTULA, **RI**- RESPIRATORY TRACT INFECTION**, P** – PNEUMONITIS,